How your mind can beat the power of food

Maastricht University

Inaugural address

Prof.Dr. Anne J. Roefs

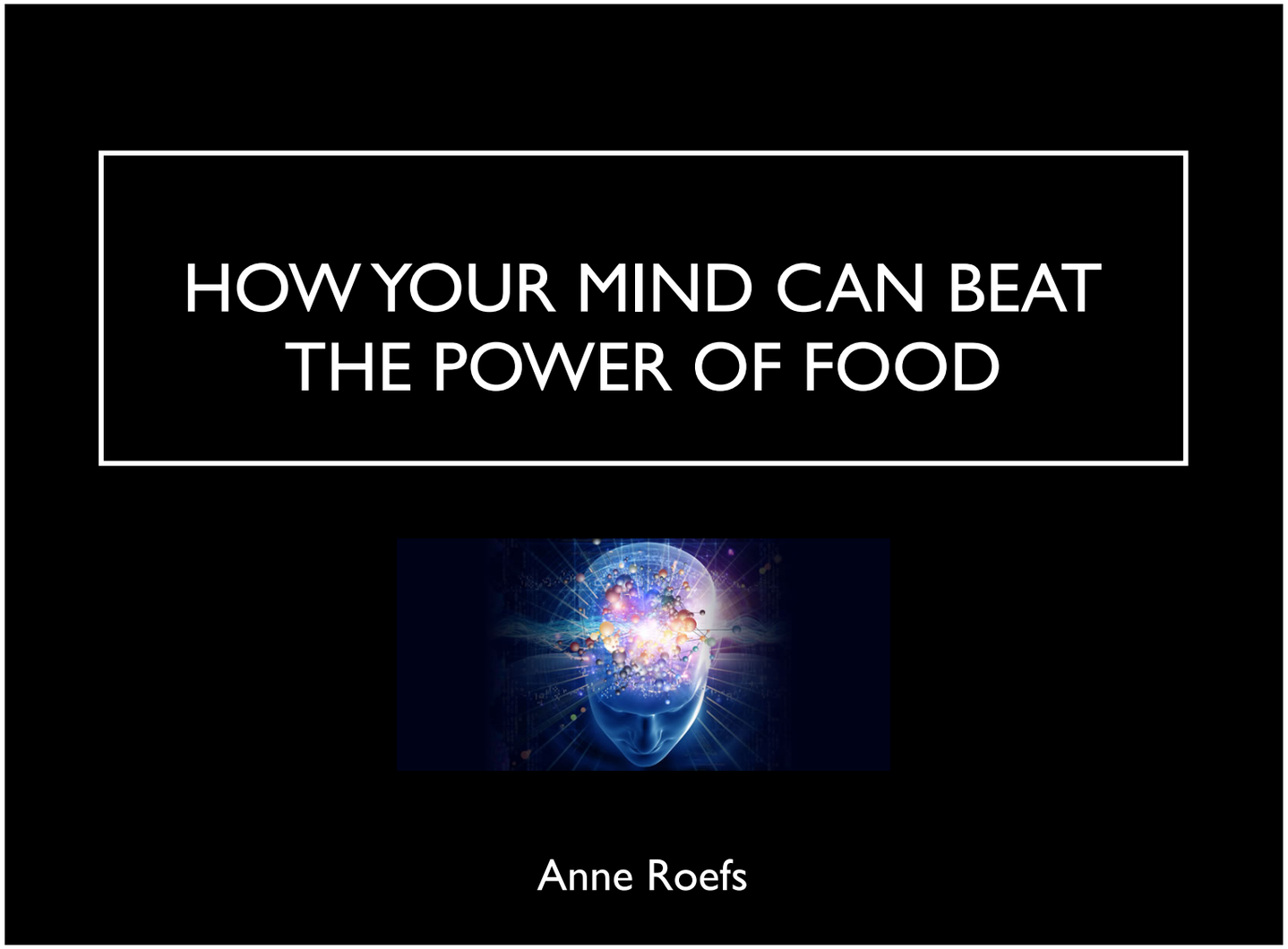
Faculty of Psychology and Neuroscience

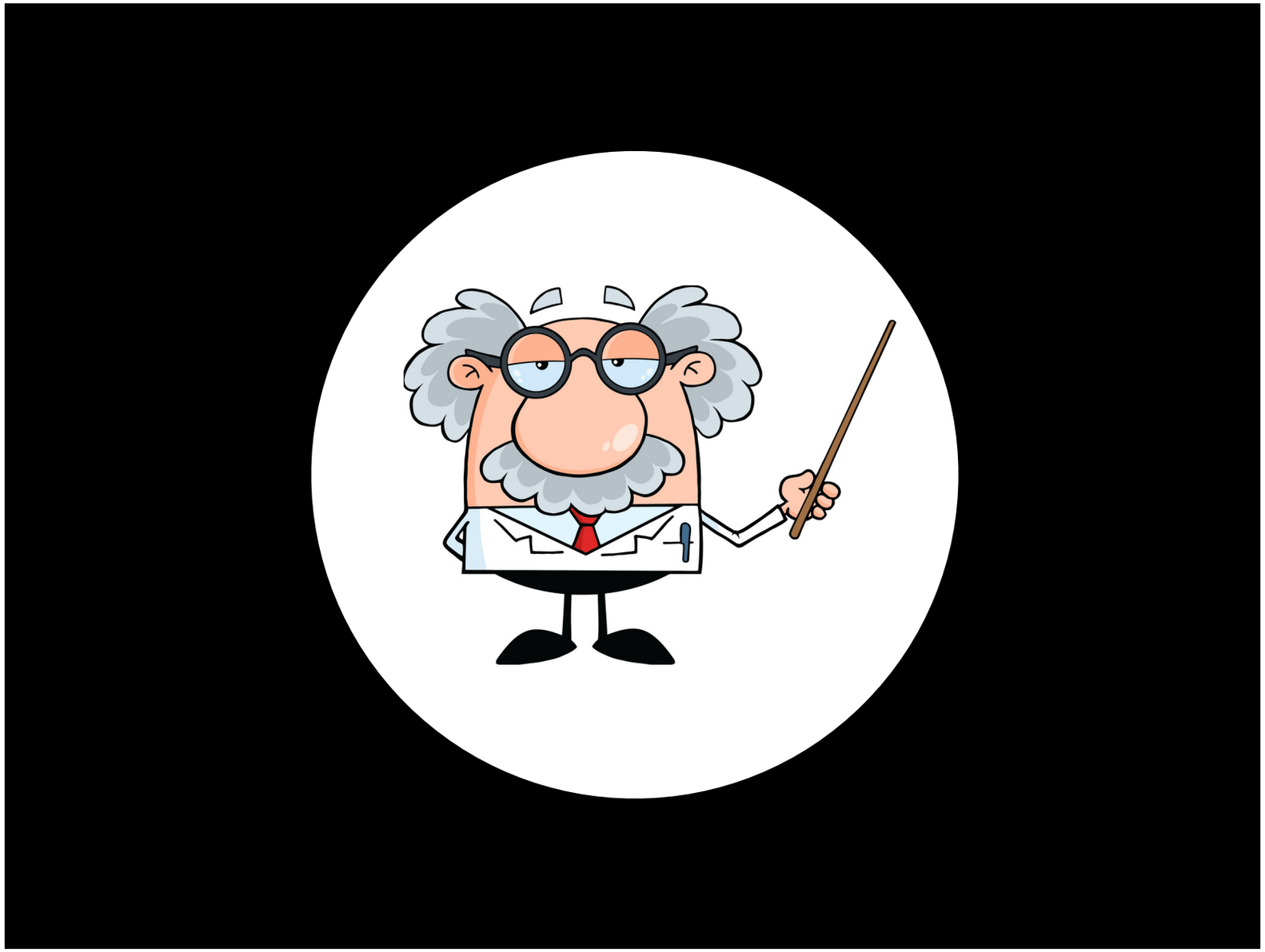
How your mind can beat the power of food

Anne Roefs

Inaugural address

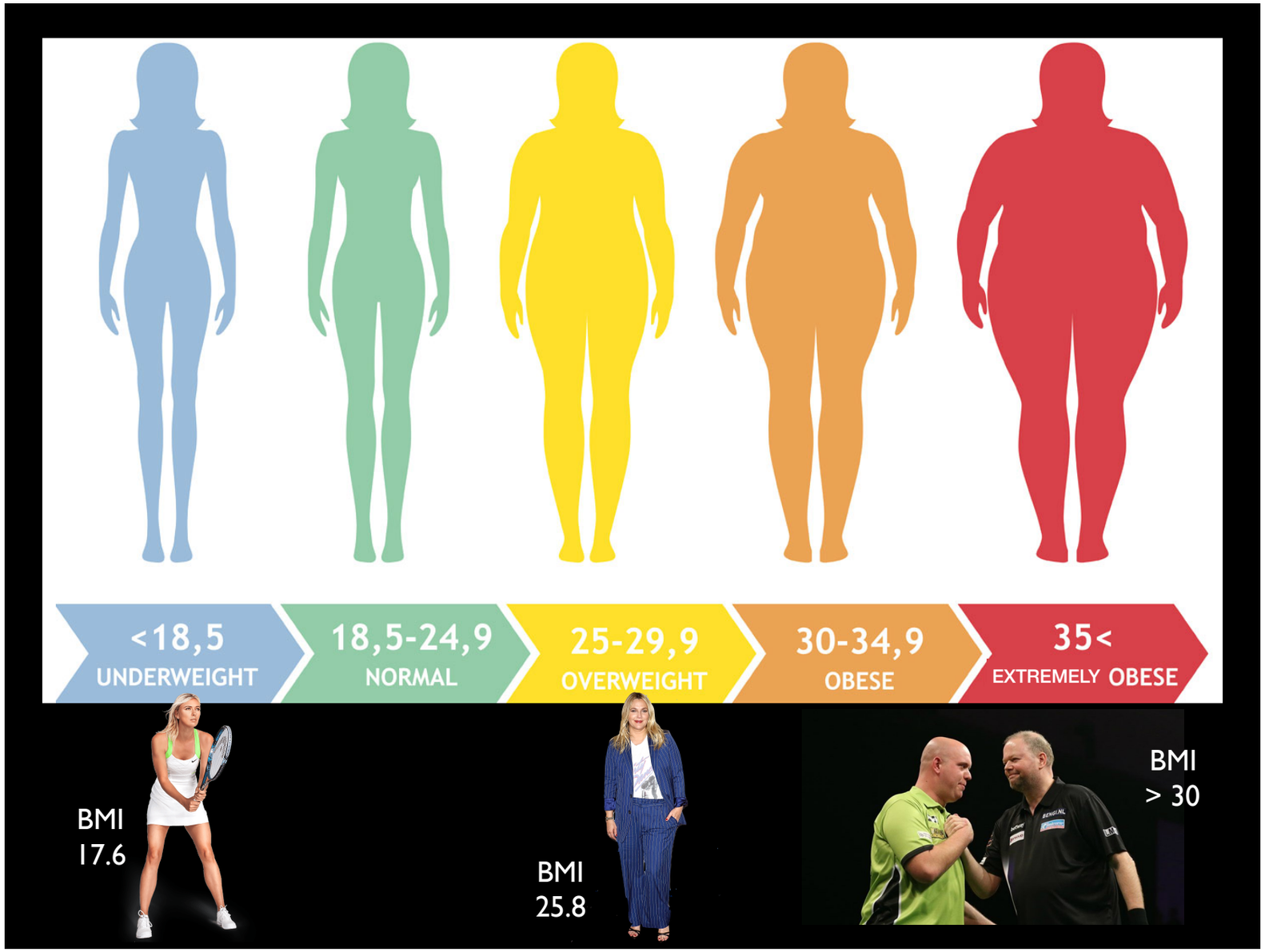
June 14, 2019 |
Maastricht University



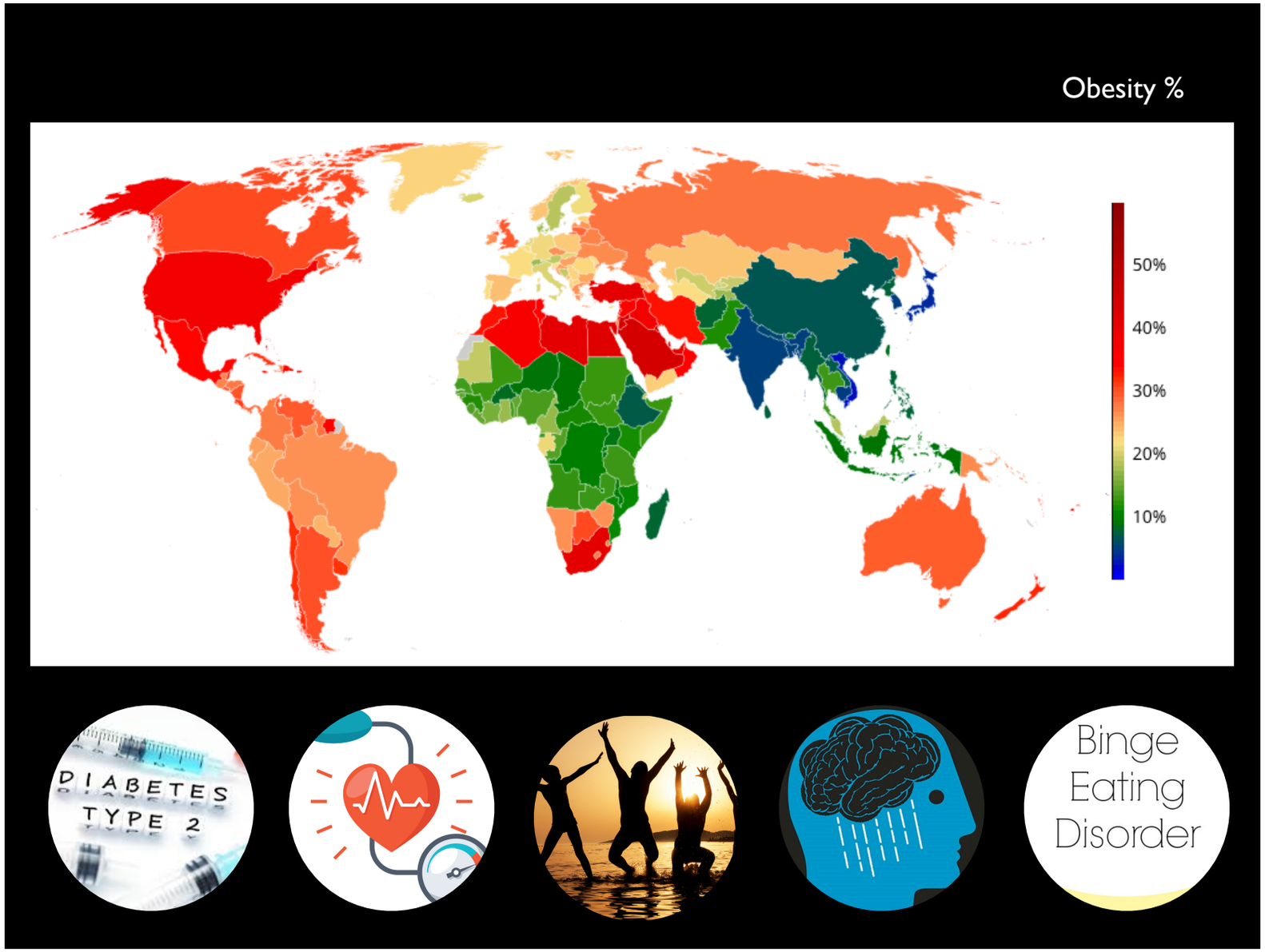


About four months ago, I read a column in Volkskrant, which was during the time I started thinking about my inaugural lecture, and was trying to get used to the idea of being a professor. The writer of this column, Professor Marleen Kamperman (Volkskrant, 2019), expressed my feelings very well. She wrote that "*she was haunted by doubts: Am I a professor? Is there no misunderstanding? Professors are knowledgeable people who know what they are doing. They have a good overview, have answers, and can explain on Radio 1 how stuff works.*"

This may sound like 'humble bragging', a statement in which one pretends to be modest, but which one is really using as a way of telling people about one's success. But, rest assured, that was not the point. I agree with Professor Kamperman, that "*admitting to not having all the answers, and having a lot of questions is the essence of science. Realizing that one doesn't know much and that one is reaching into the dark. The only thing one can do is to approach that reaching into the dark in a clever way.*" My lecture today will be about my puzzles and questions about the power of food, mind, and obesity, which I aim to address in the upcoming years. Of course, I will also share the progress that I made so far in trying to solve these puzzles. Oh, and by the way, I recently did explain how certain stuff works on Radio 1.

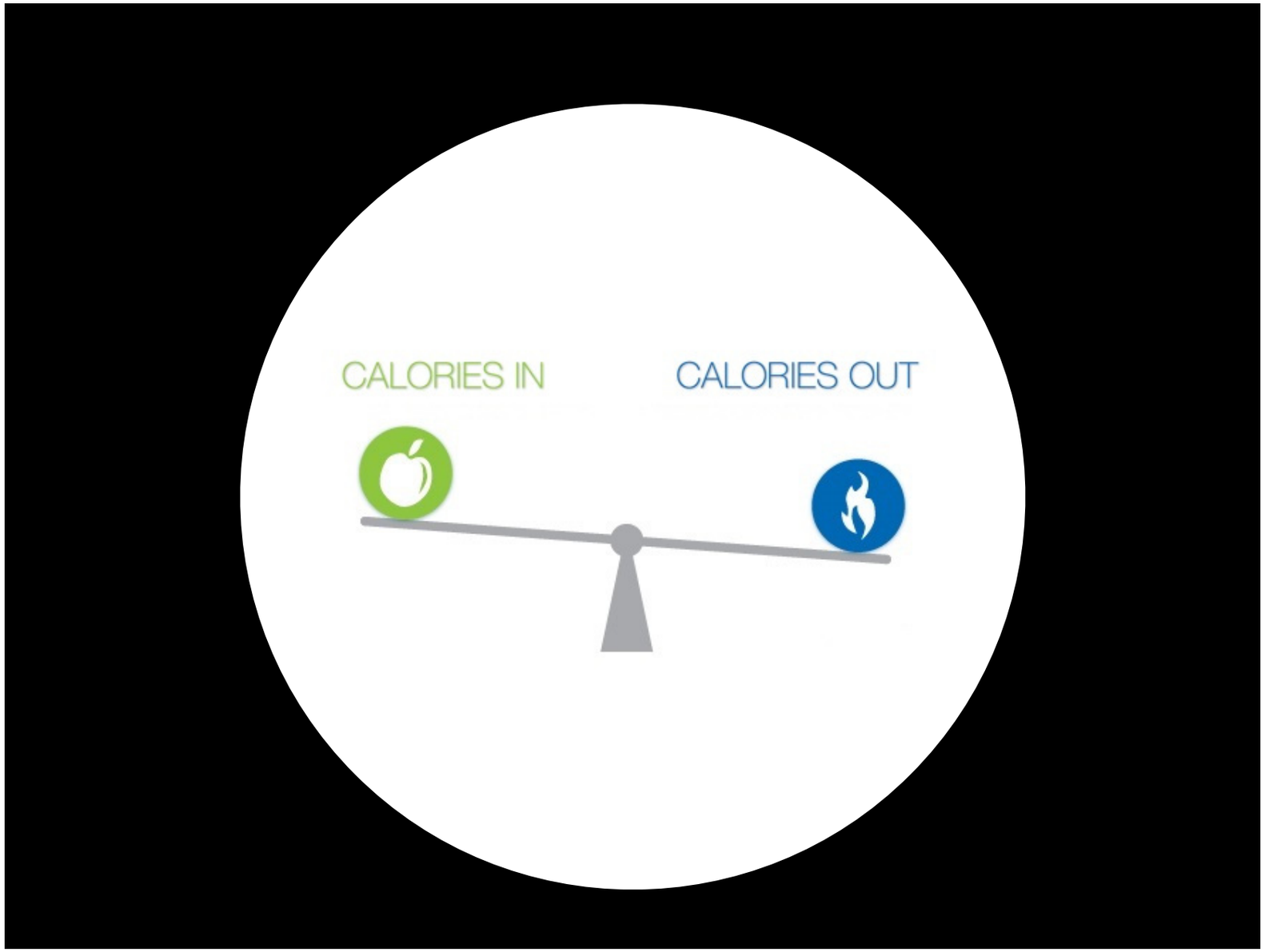


First some definitions. Overweight and obesity are typically defined by the so-called body-mass-index, the BMI, which is your weight divided by your height squared. A BMI over 25 is considered overweight, and over 30 is obese. Between 18.5 and 25 is considered a healthy weight.

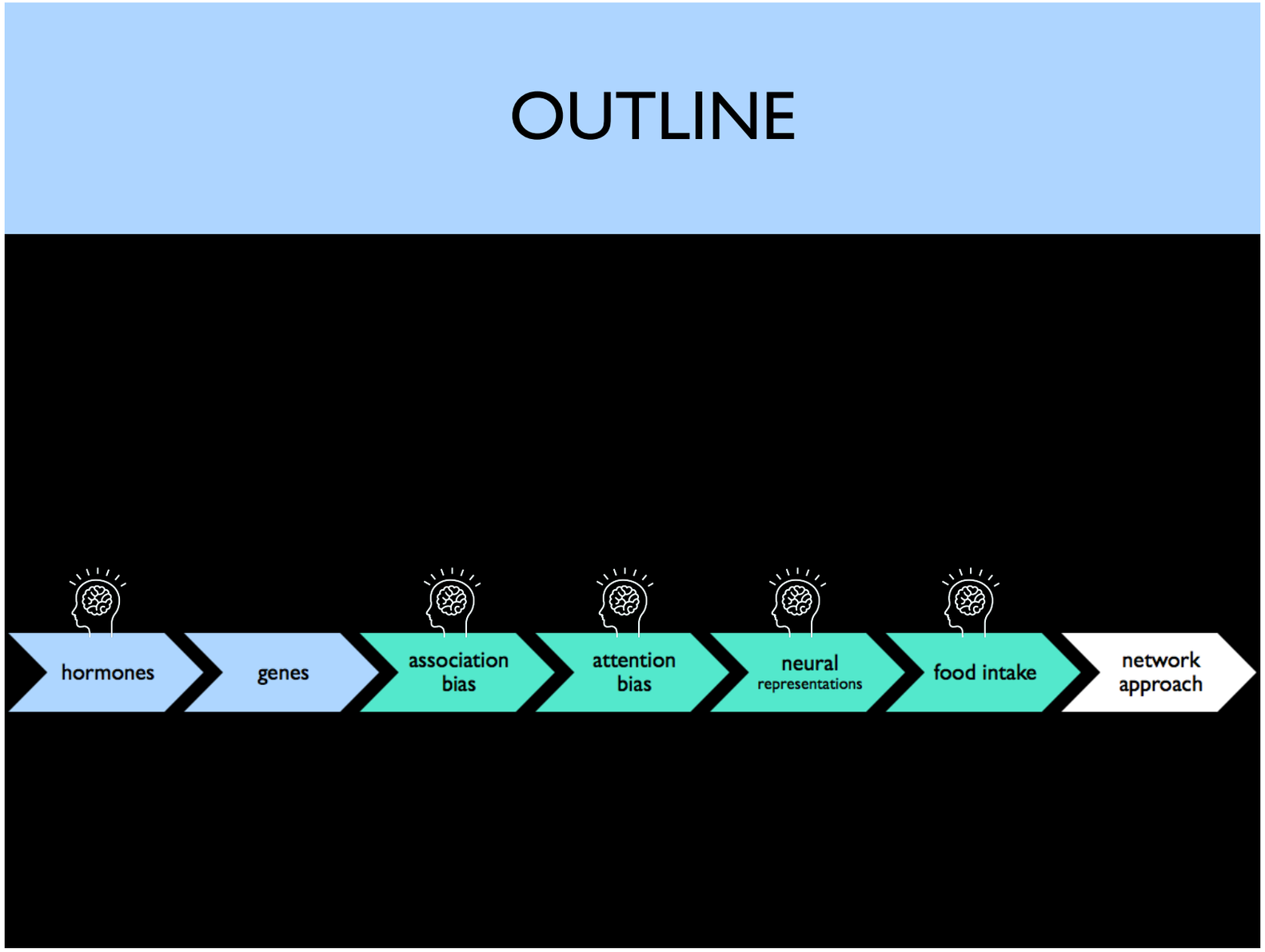


Worldwide the prevalence of overweight and obesity has increased over the last decades. It is considered an epidemic. The problem is most prevalent in for example Mexico, New Zealand, and in the USA, with about 30-35% obesity rates. In our own country the situation is not so bad, relatively speaking, with a 15% obesity rate. However, an additional 35% has a milder problem, is overweight. So, taken together, half our population is too heavy.

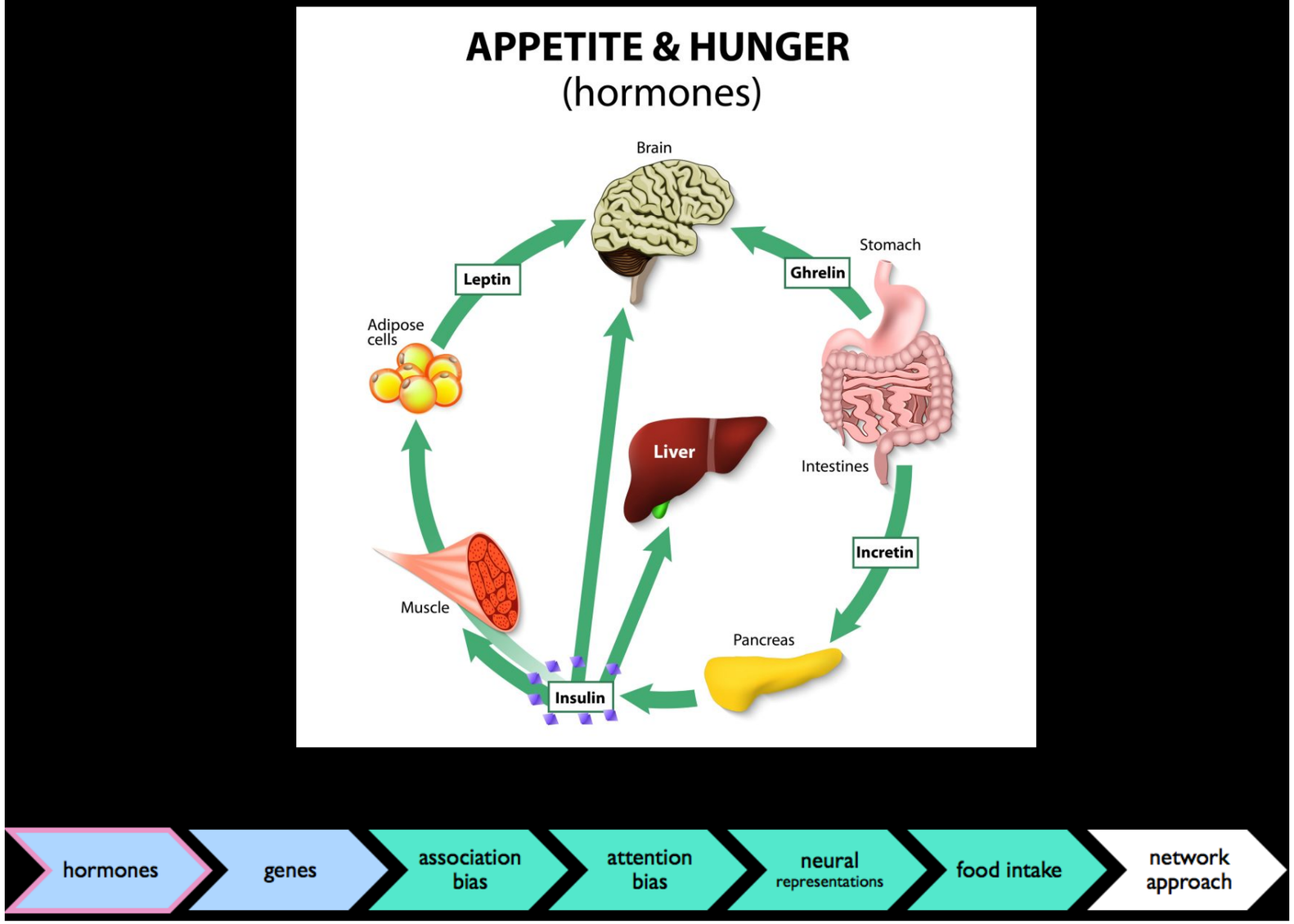
Why is that a problem? Well, being overweight or obese is associated with many negative health consequences, such as type 2 diabetes, high blood pressure, and cardiovascular problems, as well as with a reduced quality of life. Also, the risk of mental health problems – such as depressive disorder and eating disorders – is increased.



The simplest answer to the question how obesity arises is: “Obesity is the result of a prolonged positive energy balance.” More calories are consumed than are expended for an extended duration. The more interesting and difficult question is *why* energy intake and expenditure are imbalanced for so many people.



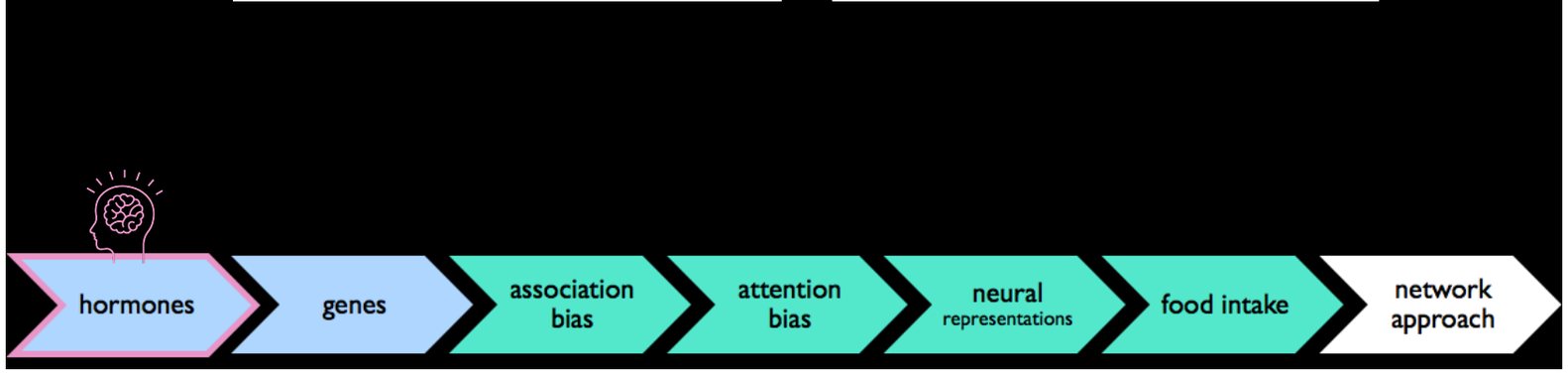
Today I will take you through research attempting to answer this question, going from hormones, to genes, to cognition, to the brain, to food intake, while highlighting the role mindset plays. At the end of the lecture, I will take it one step further, and will introduce a network approach to obesity.



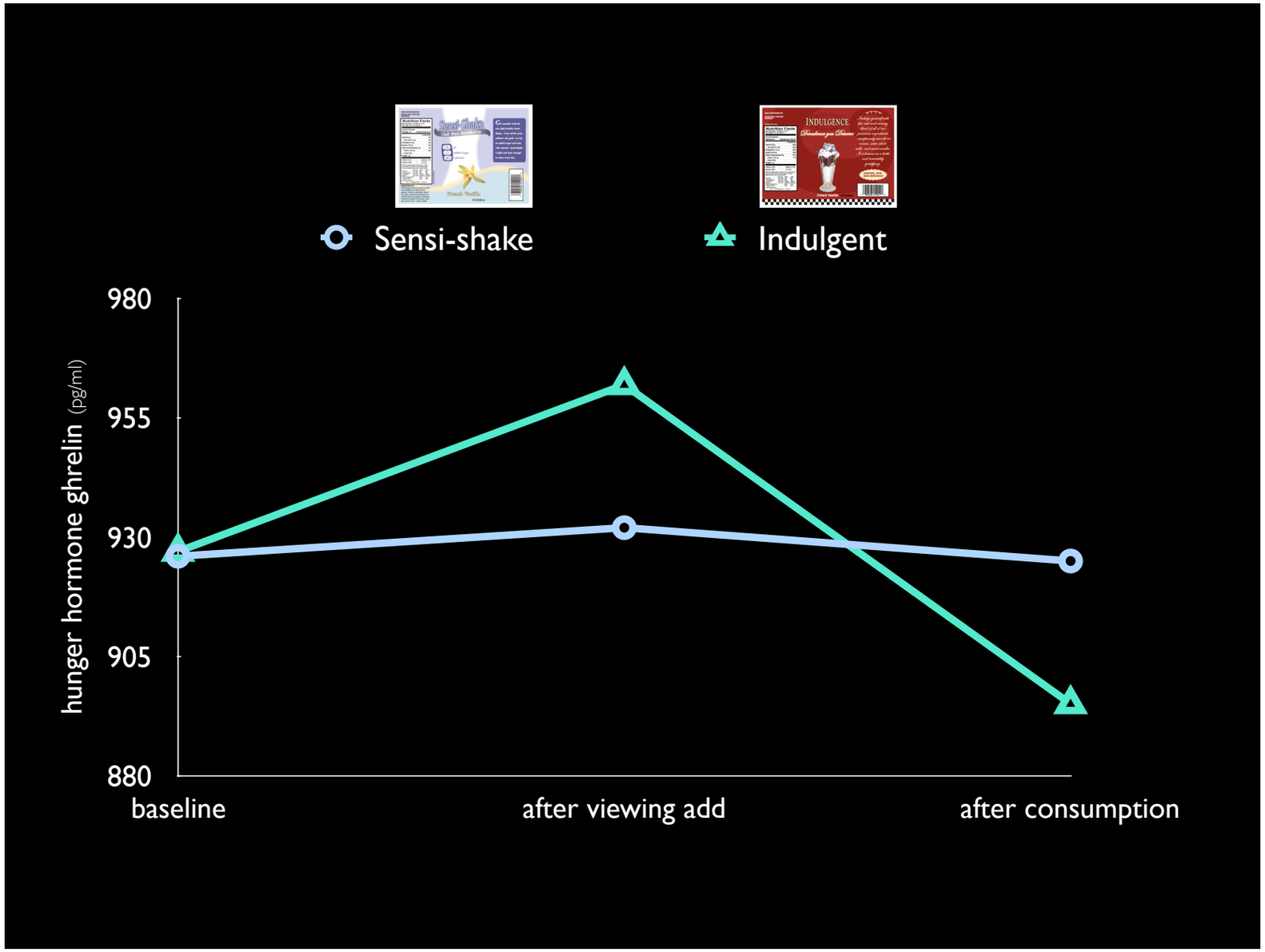
Many researchers try to understand the origins of this energy imbalance, and they often adopt a biomedical perspective, trying to find all kinds of biological disturbances contributing to a higher BMI. For example, over 30 hormones and peptides involved in the physiological control of appetite have been the subject of study over the years (Lean & Malkova, 2016), including the hunger hormone ghrelin and the satiety hormone leptin. However, this research has not resulted in a clear understanding of obesity or an effective treatment for obesity so far. For example, obese people do not seem to suffer from a too low concentration of the satiety hormone leptin. Moreover, the injection of leptin in obese adults, after a 3-week diet, did not lead to significantly more weight loss as compared to placebo (Zelissen et al., 2005). Taken together, the consensus seems to be that hormonal disturbances do play a part, but can never fully explain the obesity epidemic, and there is much unclear about whether these disturbances are causal or secondary to obesity (Lean & Malkova, 2016).

Crum et al. (2011). *Health Psychology*

380 kcal vanilla milkshake



Moreover, is this hormonal control of appetite really a closed-loop system, only affected by energy intake and expenditure? Maybe not! Here's the first evidence in this lecture on how one's **mind** can influence how one's body responds. Crum and colleagues (2011) studied how only thinking that one is satiated affects the ghrelin – the hunger hormone – response of one's body. In this study, participants came to the lab twice. Each time, they received a 380-kcal vanilla milkshake, which they fully consumed. One time they were led to believe that they were drinking an 'indulgent' milkshake, something to enjoy. The other time, they were led to believe that they were drinking a sensi-shake, a healthy drink. The level of the hunger hormone ghrelin was measured at baseline, after watching the advertisement, and after consumption.

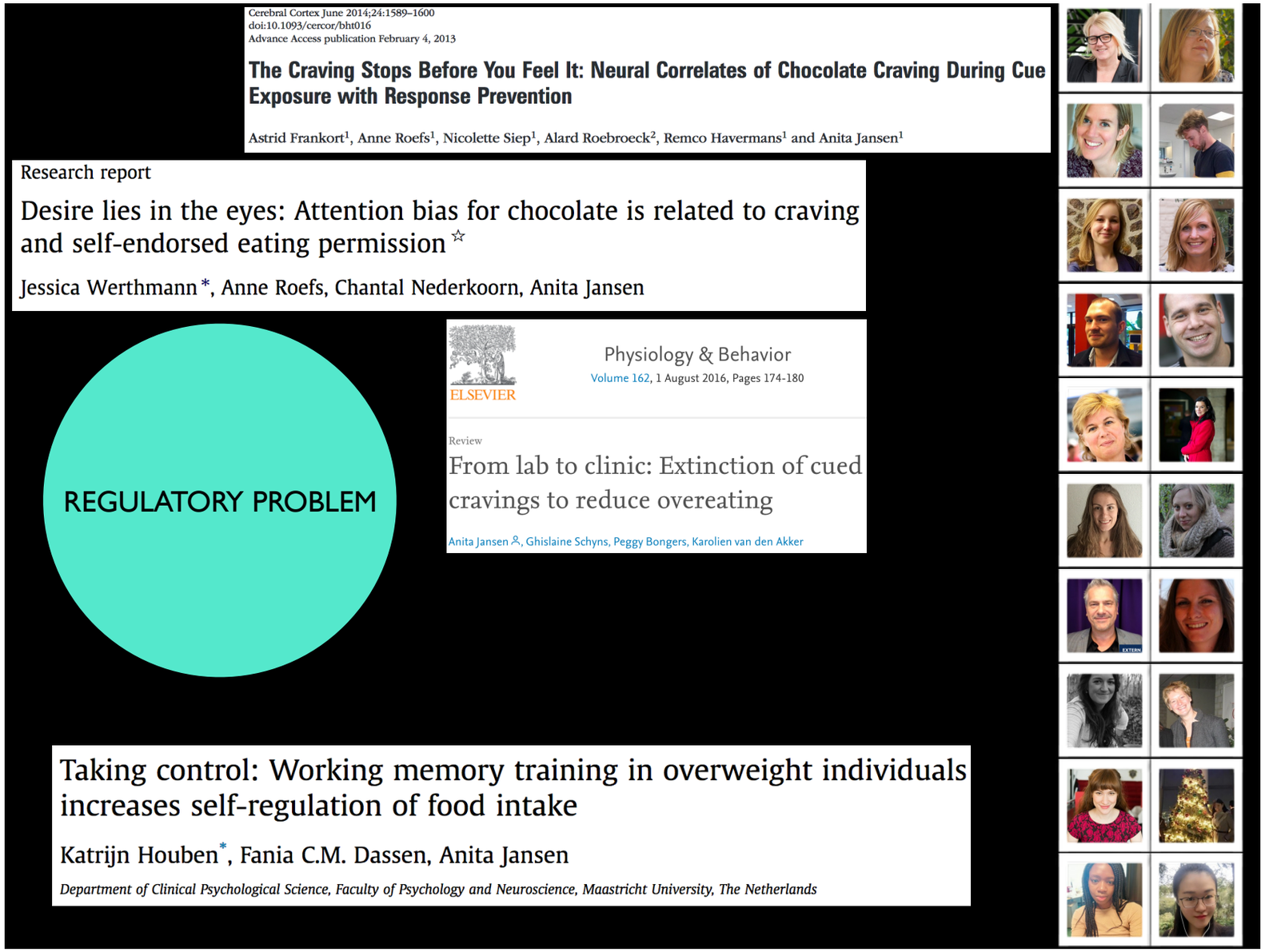


In the sensi-shake condition, the hormonal response was almost flat, whereas in the indulgent milkshake condition, the level of the hunger hormone ghrelin rose from baseline to advertisement, and it dropped after consumption. So, the hunger hormone ghrelin responded differently when participants *thought* they were receiving a high-caloric milkshake as compared to when participants *thought* they were receiving a low-caloric milkshake. So, one's mind affects biology.

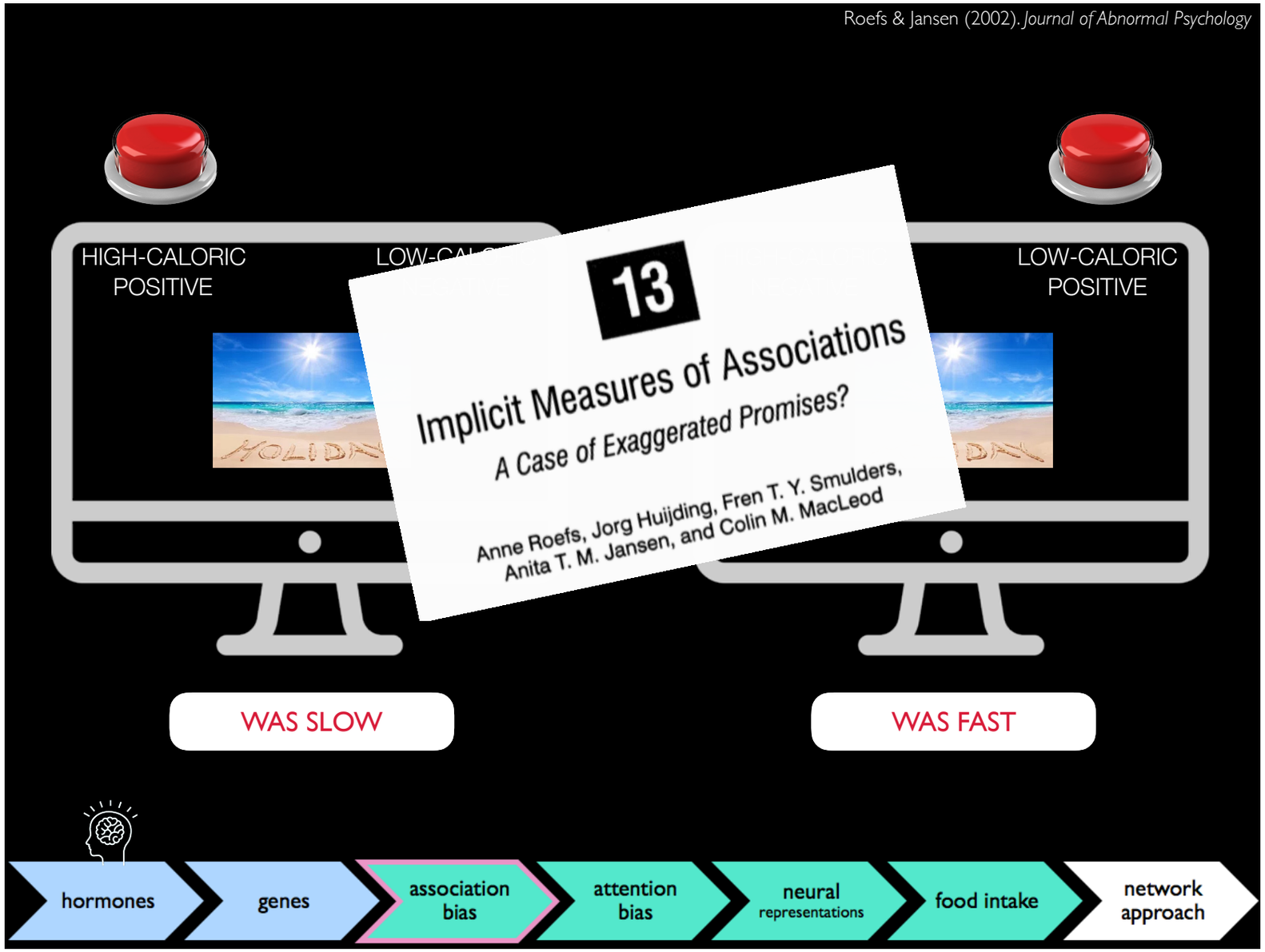
Another biomedical factor that has received much attention is genetics. Indeed, about 67% of the variability in BMI has a genetic basis (Ravussin & Bogardus, 2000). For most obese people, it is not simply one obesity-gene though, the genetic risk is spread across probably hundreds of genes. The more high-risk genetic variants one has, the more likely one is to become overweight (Khera et al., 2019). But, genetics does not mean that nothing can be done about it, one does not simply inherit a high BMI. This is how my youngest daughter does seem to think how genetics works. Recently, she just wouldn't listen, and her defense was: "I cannot help that, I was just born this way." Like my daughter can of course learn to listen, people with a risky genetic profile are not doomed.

A person who is genetically predisposed to develop obesity, would not have a problem if he lived on an island on which only fruits and vegetables were available, and physical activity was a necessary part of daily life. Our environment has changed quite dramatically though over the last century. In our current Western environment, we are surrounded by palatable high caloric foods, and in our daily lives there is not much need for physical activity. This so-called obesogenic environment makes it hard for people to stay or become lean. This is especially true for people with a genetic predisposition. They do not inherit a higher BMI directly, but inherit a regulatory problem, a responsive appetite system (Konttinen et al., 2015; Llewellyn & Fildus, 2017; Llewellyn & Wardle, 2015; Wardle, 2009). This could for example take the form of genetically determined preferences for certain types of food or for less physical activity.

After I explained my youngest daughter a bit about nature and nurture, she replied: "But then it means that you just didn't raise me well, and that you need to do better." I gave her a kiss, and I think she learned a lot that night.



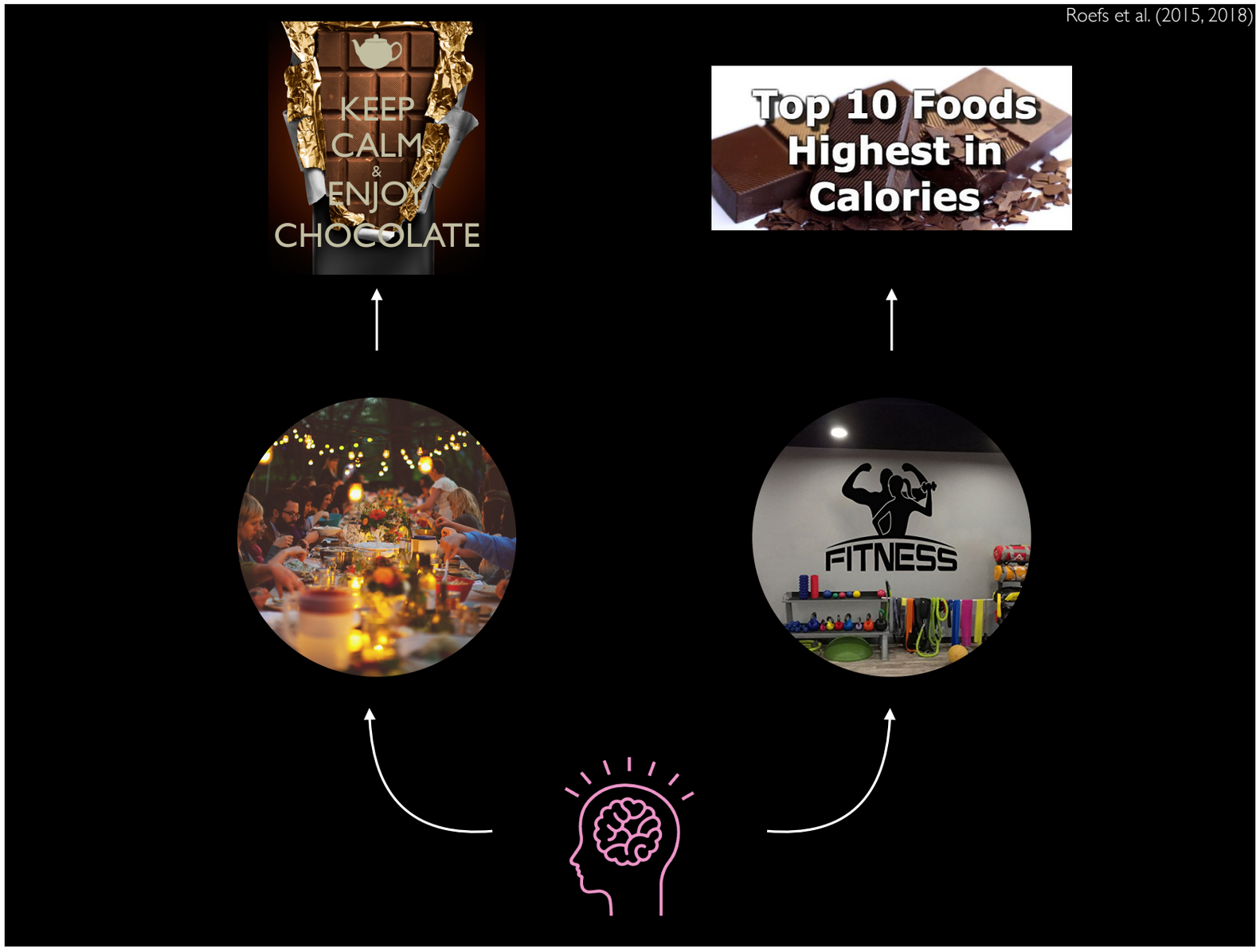
Exactly this regulatory problem has been the focus of much of my research over the past years, as well as from the researchers in our group. Today I will introduce some of that research to you.



My own research on this topic started during my PhD back in 2000-2004, where I addressed the question whether obese people display an increased positive association with high-caloric foods. I used response-latency based paradigms, such as the Implicit Association Test and the affective priming paradigm to learn about the strength of associations that come to mind when viewing high-caloric foods. Today I do not have time to explain these paradigms in detail, but in essence these paradigms require participants to respond as quickly as possible to visual stimuli presented on a computer screen, and the idea is that participants respond faster when presumably associated concepts, such as 'high-caloric and 'positive, are paired than when presumably non-associated concepts, such as 'low-caloric and 'positive are paired.

Quite unexpectedly, obese people associated high-caloric foods more strongly with negative than with positive, as compared to lean people. (Roefs & Jansen, 2002). Though it was the exact opposite of what we had hypothesized, this negative association bias makes sense if one considers the typically large number of dieting attempts that obese people have undertaken. Chocolate is not only tasty, but also represents a ton of calories. Though the assumption of these paradigms is that so-called automatic or implicit attitudes are assessed, and that these should be positive for craved substances, the story is not so simple.

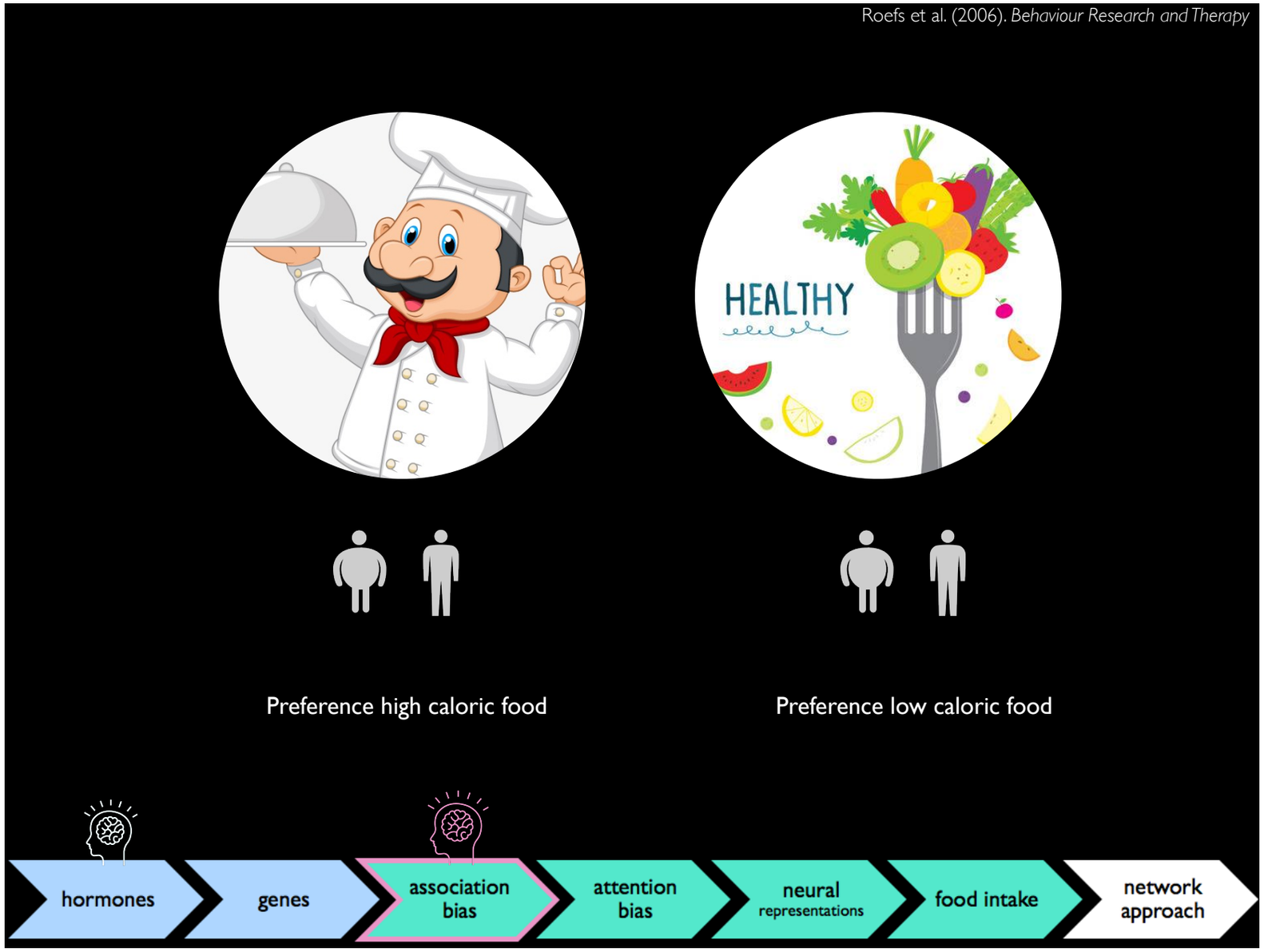
Until now, much research has assumed that overweight people tend to *firstly* and *automatically* focus on the hedonic aspects of high-caloric foods, on how much they like the taste of these foods (Hofmann, Friese, & Strack, 2009; Smith & DeCoster, 2000; Strack & Deutsch, 2004), and that therefore high-caloric foods should always elicit positive associations and attract attention.



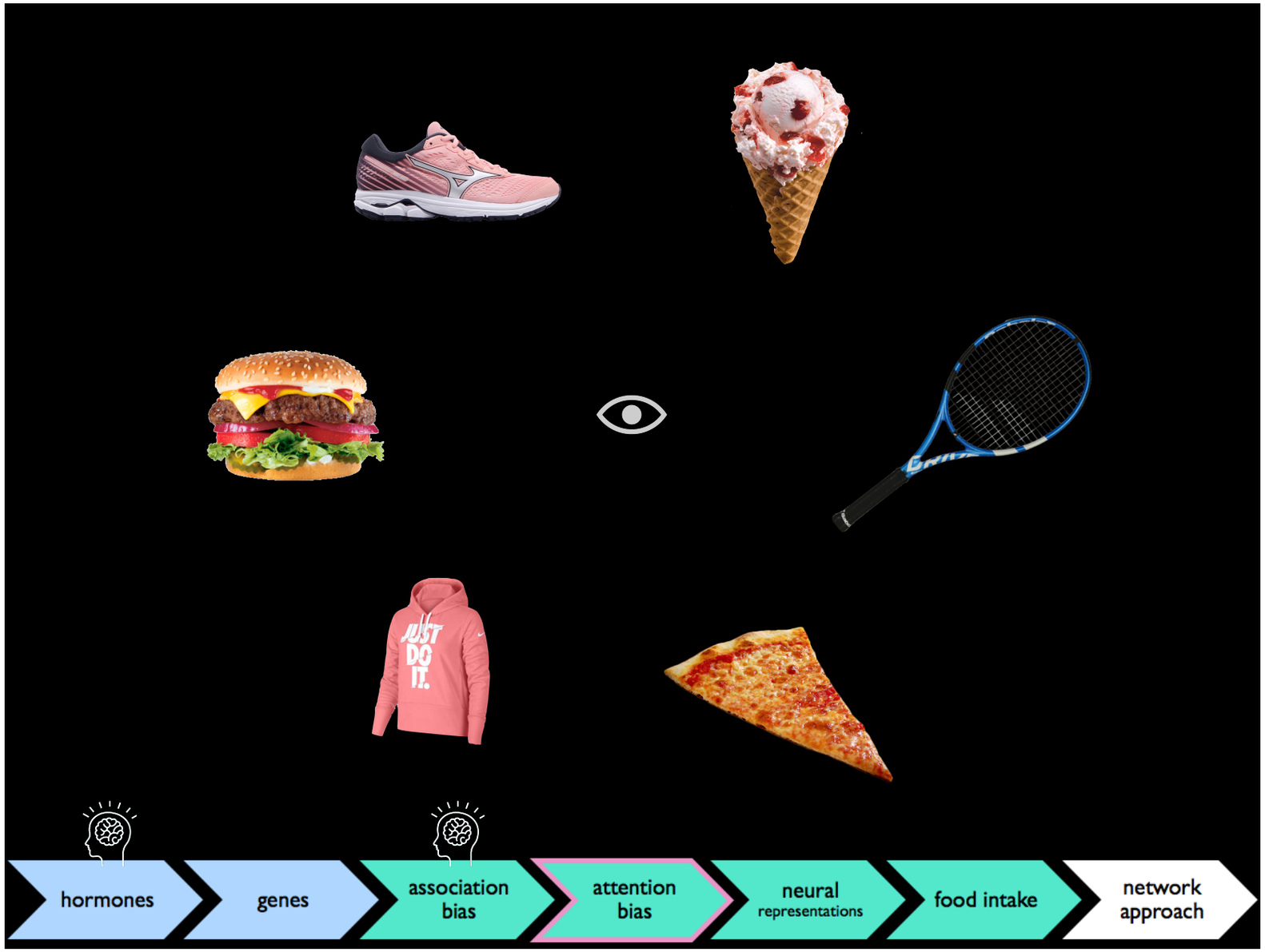
The central idea that I am currently investigating is that hedonic value does *not* always take precedence, but that it is a matter of mindset, for any person. Just imagine, how would you view chocolate while you are at a dinner party? And how would you view chocolate when you just walked by a fitness gym?

High-caloric food perception has a double-sided nature: High-caloric foods often have a high hedonic value and at the same time these foods have a low health value because their overconsumption contributes to weight gain (Roefs et al., 2015, 2018).

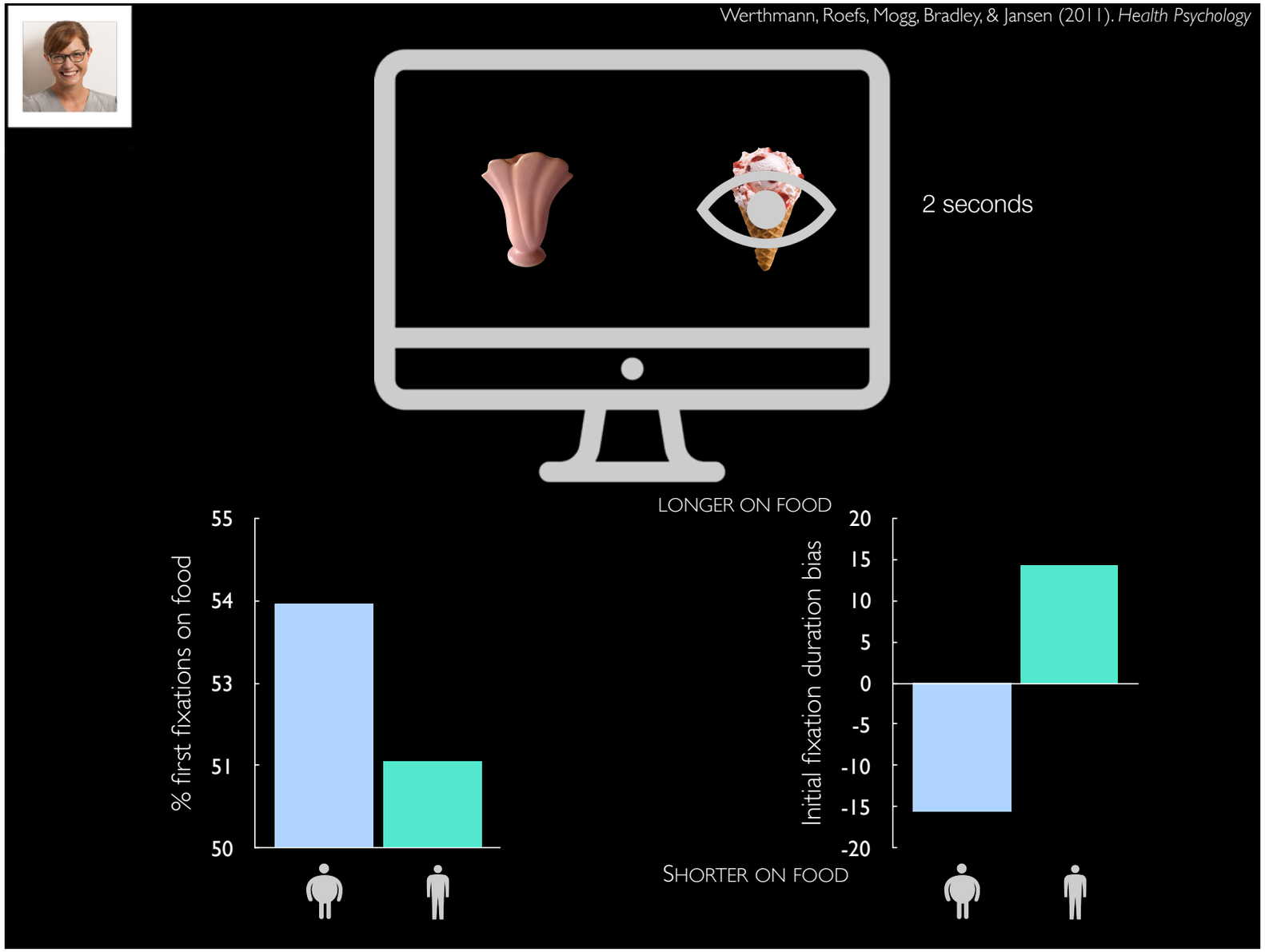
So, people's *mindset* may alternate between focusing on hedonic versus health value, depending on for example their emotional or physiological state and the current situation or context. Importantly, mindset may vary—unbeknownst to the researcher—within and across participants as well as within and across studies, complicating the interpretation of study findings.



Also at the time of my PhD, I thought about how one's **mind** can affect the power of food, and I manipulated participants' mindset just before measuring their association-bias with food. Half of the participants were asked to imagine they were a chef in a restaurant about to prepare a nice dinner for a wedding, whereas the other half were given information on healthy eating habits. In each of these groups, half of the participants were obese, and the other half were healthy-weight. The manipulation of mindset fully determined the associations with food: In the restaurant condition a preference for palatable and high-caloric foods was observed, whereas in the health condition a preference for healthier foods was observed. Body weight did not affect these associations at all (Roefs et al., 2006).



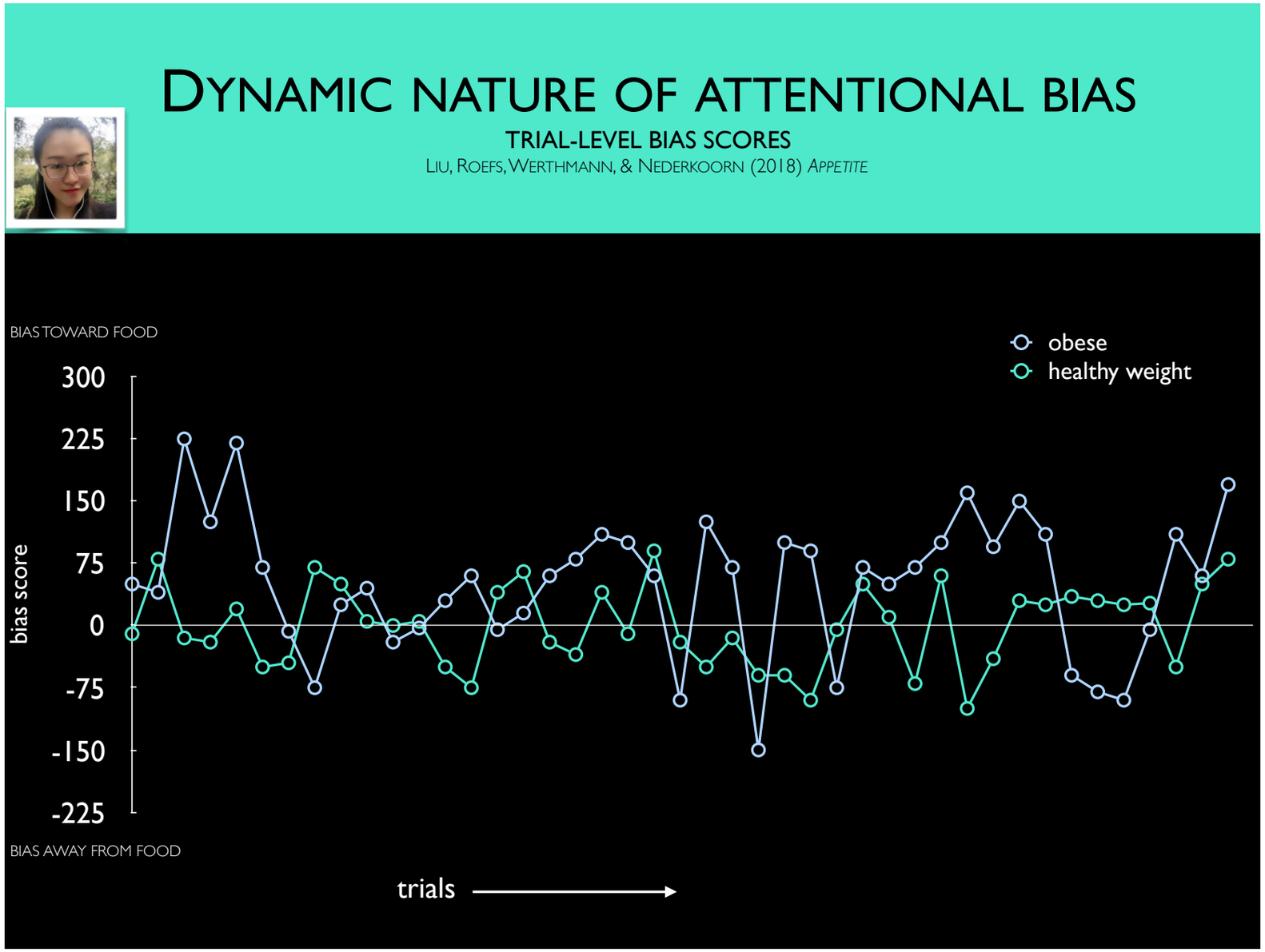
One other way our obesogenic environment may affect people differently, is that food may not attract an equal amount of attention in all people. In this example, some people's attention may be preferentially drawn to pizza, hamburgers, ice-cream, whereas other people's attention may be drawn to tennis rackets, running shoes, or clothes. The idea is that if one's attention is drawn preferentially to high-caloric palatable foods, craving will increase, and food intake will become more likely. In its turn, an increased level of craving may heighten attention for food, and the self-perpetuating circle is complete (Field et al., 2016).



My former PhD-student Jessica Werthmann and I designed an experiment to test the idea that obese people's attention would be preferentially drawn to high-caloric food stimuli and attention would also remain on these stimuli longer as compared to lean people (Werthmann et al., 2011). On each trial, a high-caloric food stimulus was presented alongside a neutral visually matched control stimulus for 2 seconds. We measured the direction of the first eye-movement, the duration of the initial fixation and total time of looking. We found that overweight participants directed their first eye-movement more often toward high-caloric foods than lean people, but subsequently showed reduced maintenance of attention on these food stimuli. So, they displayed an approach-avoidance pattern of attention.

At the end of Jessica's PhD, we reviewed all scientific literature addressing attentional bias for high caloric food in obesity (Werthmann et al., 2015), and the results across studies appeared to be highly variable, with evidence supporting attentional approach (e.g., Castellanos et al., 2009), attentional avoidance (e.g, Nummenmaa et al., 2011), and attentional approach-avoidance (e.g., Werthmann et al., 2011) of high-caloric foods, and also studies finding no differences at all between overweight and lean people (e.g., Loeber et al., 2012).

Also, for attentional bias, we proposed that mindset may be a crucial factor. We currently test this idea in obese and lean people, but we already have some evidence how mindset moderates attentional bias for high-caloric foods in high and low restrained eaters. That is, people with or without the intention to limit their food intake. We induced either a hedonic or a health mindset, and measured attentional bias for high-caloric foods. For low-restrained eaters, the mindset induction had no effect. For high-restrained eaters, we found that high-caloric foods stopped drawing attention when they had a health mindset (Werthmann et al., 2016). So, the mind can beat the attention-grabbing powers of food.

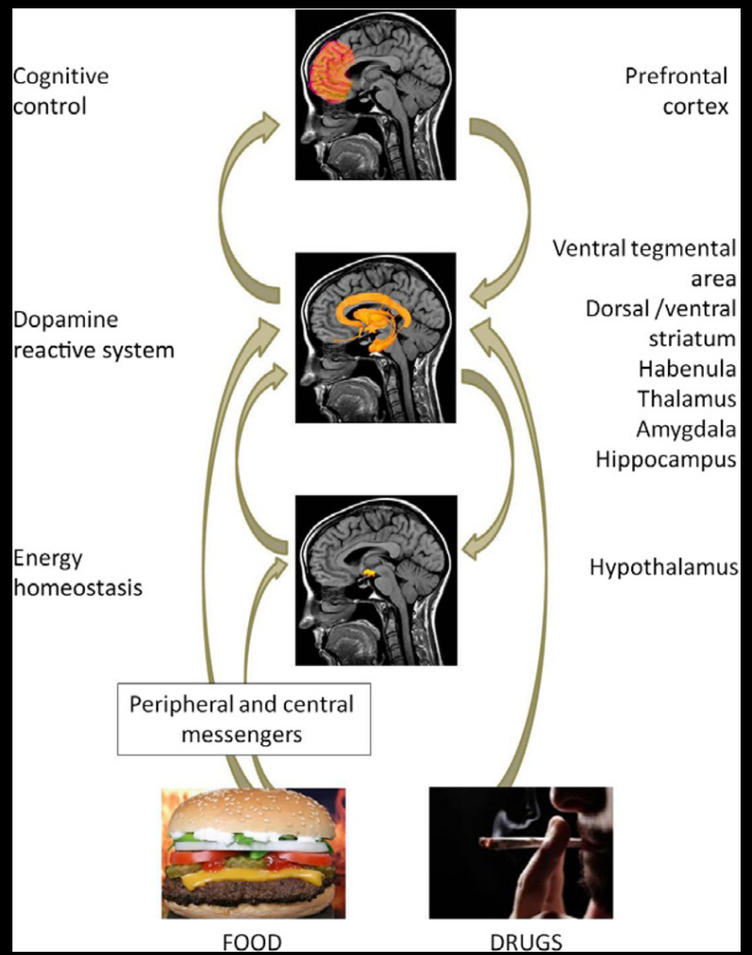


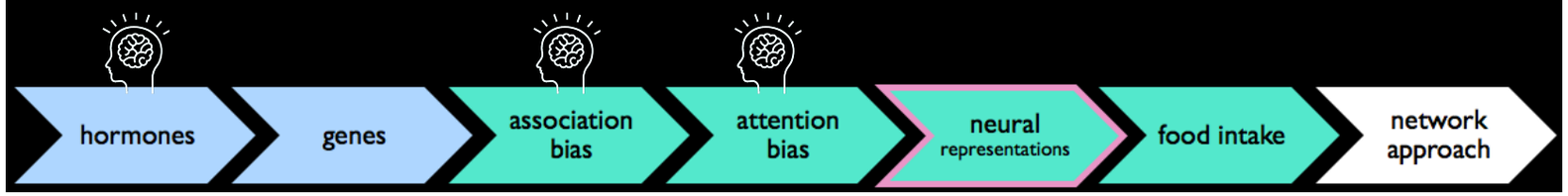
More recently, our PhD student Yu Liu re-analyzed some datasets of Jessica Werthmann, to test the dynamic nature of attentional bias for food (Liu et al., 2019). In all attentional bias studies, all trials of the same type are averaged together, which hides how attention may fluctuate during the task. By computing trial-level-bias-scores (TLBS), one can study exactly that, how attentional bias fluctuates over trials during the experiment. As can be seen in this graph, attentional bias for food fluctuated over trials, with evidence for attention towards and away from foods. When combining datasets of three studies, we found that this variability in attention bias for food was related to a higher BMI. So, attention for food fluctuated more in people with a higher BMI.

A puzzle for the future is, what exactly caused this higher variability. Another idea that we will test is whether attentional variability will decrease when a hedonic or health mindset is induced.

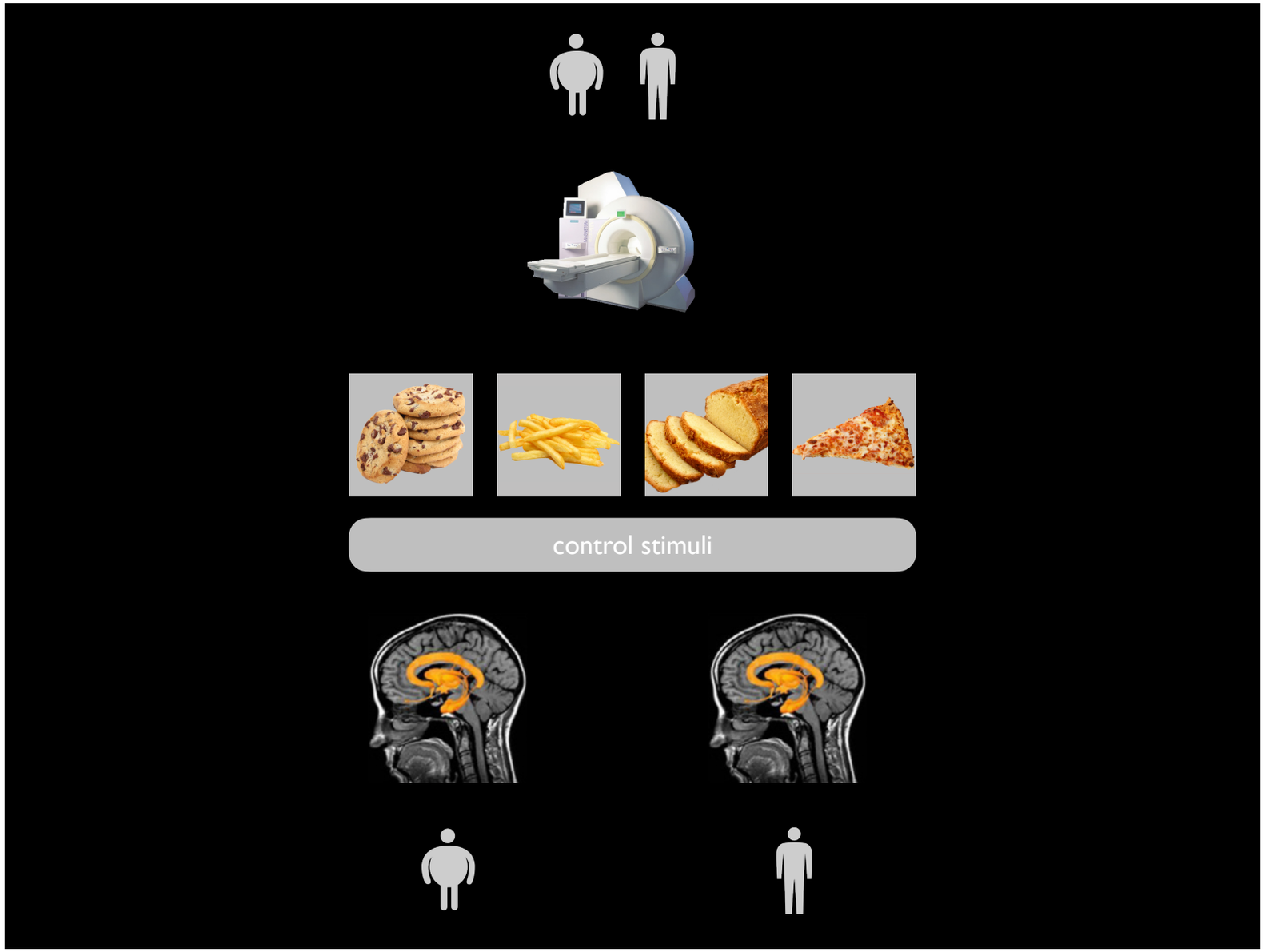
OBESITY AND EATING DISORDERS INVOLVE IMBALANCES IN THE REWARD AND SELF-CONTROL CIRCUITS OF THE BRAIN (P. 14). THE DISRUPTION OF BRAIN DOPAMINE PATHWAYS IS CENTRAL TO THESE DISORDERS.

VOLKOW ET AL. (2013)
OBESITY REVIEWS





In a related research line, I am searching for differential neural responses to food that may explain the differential vulnerability to our obesogenic environment. In the literature it is stated that “obesity and eating disorders involve imbalances in the reward and self-control circuits of the brain (Volkow et al., 2013; p. 14)”, and that the disruption of brain dopamine pathways is central to these disorders (Volkow et al., 2013). Specifically for obesity, the idea is that the anticipatory neural response to food in the reward circuit is increased, whereas the consummatory neural response is reduced. So, when viewing foods in the environment, craving levels would be higher in obese people, and that would then be reflected in an increased anticipatory neural response to these food cues. The reward experienced from actual food intake would be less, and therefore the consummatory neural response would be lower in obese than in healthy-weight people. Obese people would have to consume more food to achieve the same level of neural reward responding, and are more enticed by food cues in their environment.



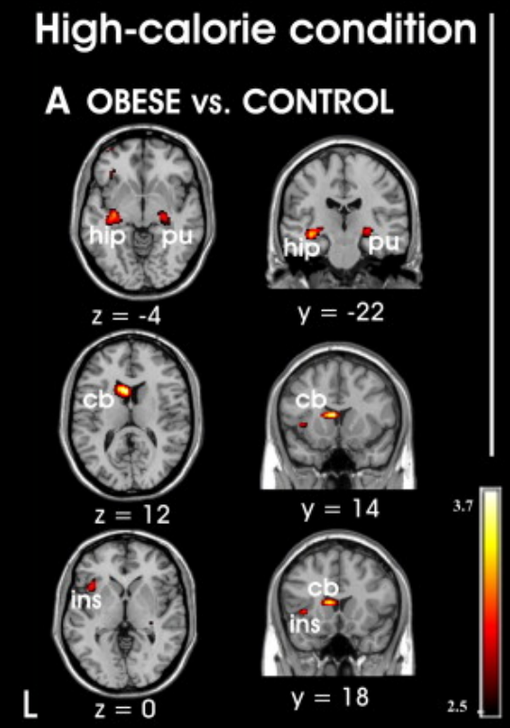
Quite some studies have addressed this anticipatory neural response in obese people, and typically participants view pictures of high-caloric palatable foods and some kind of neutral control pictures. Often a passive-viewing paradigm is used in which participants are merely instructed to look at the pictures while in the MRI scanner. Using functional Magnetic Resonance Imaging, fMRI, a measure of neural activity is obtained. The idea is here that the level of activity in reward-related regions of the brain is proportionate to the reward-value of presented stimuli, and this should be higher in obese people. What is the evidence for this idea?

* icon by Daniel Behrends (male icon) from thenounproject.com.

EXAMPLE 1

ROTHEMUND ET AL. (2007). *NEUROIMAGE*

FOOD STIMULI ACTIVATE REGIONS RELATED TO **REWARD ANTICIPATION** (STRIATUM) AND **TASTE REPRESENTATION** (INSULA) MORE STRONGLY IN OBESE PEOPLE.



Increased activation in:

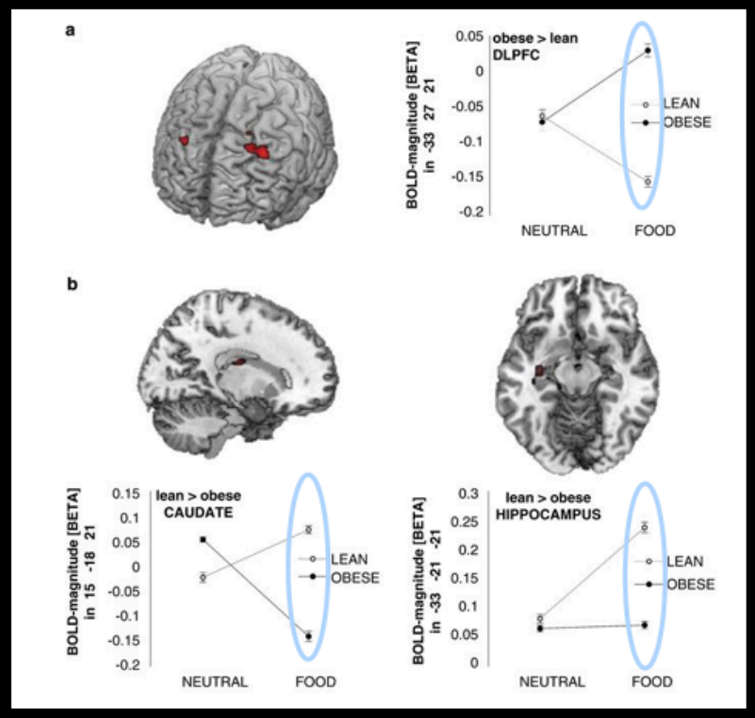
- dorsal striatum (putamen and caudate)
- insula
- hippocampus

Rothemund and colleagues (2007) compared the neural response to pictures of high-caloric foods in obese and lean people. They observed increased neural responding in the striatum, the hippocampus, and the insula. Their interpretation is that food stimuli activate regions connected to reward anticipation and habit learning (striatum) and taste representation (insula) more strongly in obese people. So far so good, but here's another example.

EXAMPLE 2

DAVIDS ET AL. (2010). *INTERNATIONAL JOURNAL OF OBESITY*

OBESE CHILDREN HAVE INCREASED INHIBITORY CONTROL (DLPFC), AND FOOD CUES DO NOT ACTIVATE THEIR REWARD SYSTEM AS MUCH (CAUDATE, HIPPOCAMPUS)



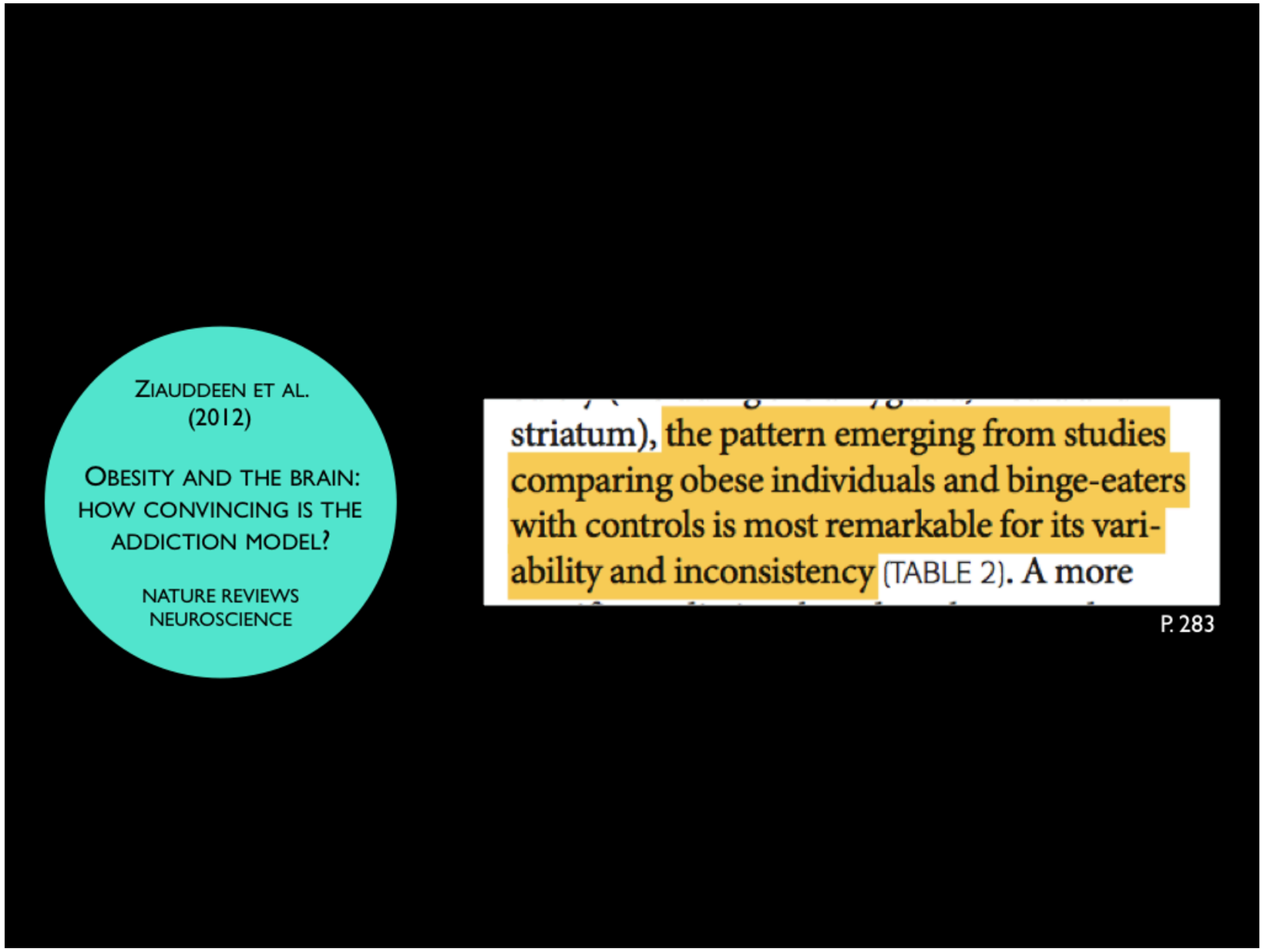
Decreased activation in:

- dorsal striatum (caudate)
- hippocampus

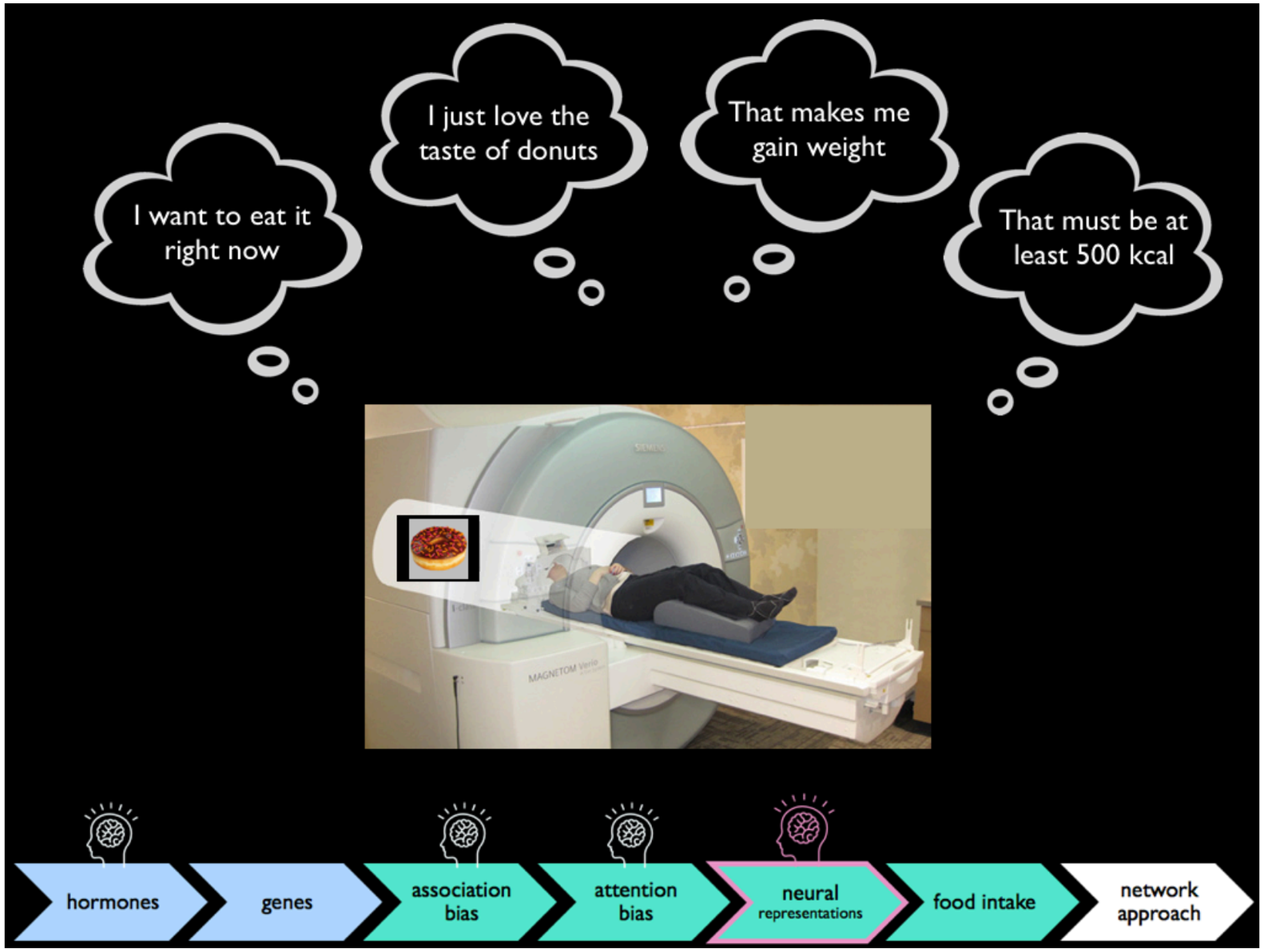
Increased activation in:

- dorsolateral prefrontal cortex (DLPFC)

Davids and colleagues (2010) compared the neural response to food versus neutral pictures in obese and lean children. Quite opposite to the previous study I talked about, they observed decreased activity in the dorsal striatum and hippocampus, observed no significant effect in the insula, and increased neural activity in the dorsolateral prefrontal cortex. Their interpretation is that obese children have increased inhibitory control (DLPFC), and food cues do not activate their reward system as much (caudate, hippocampus).

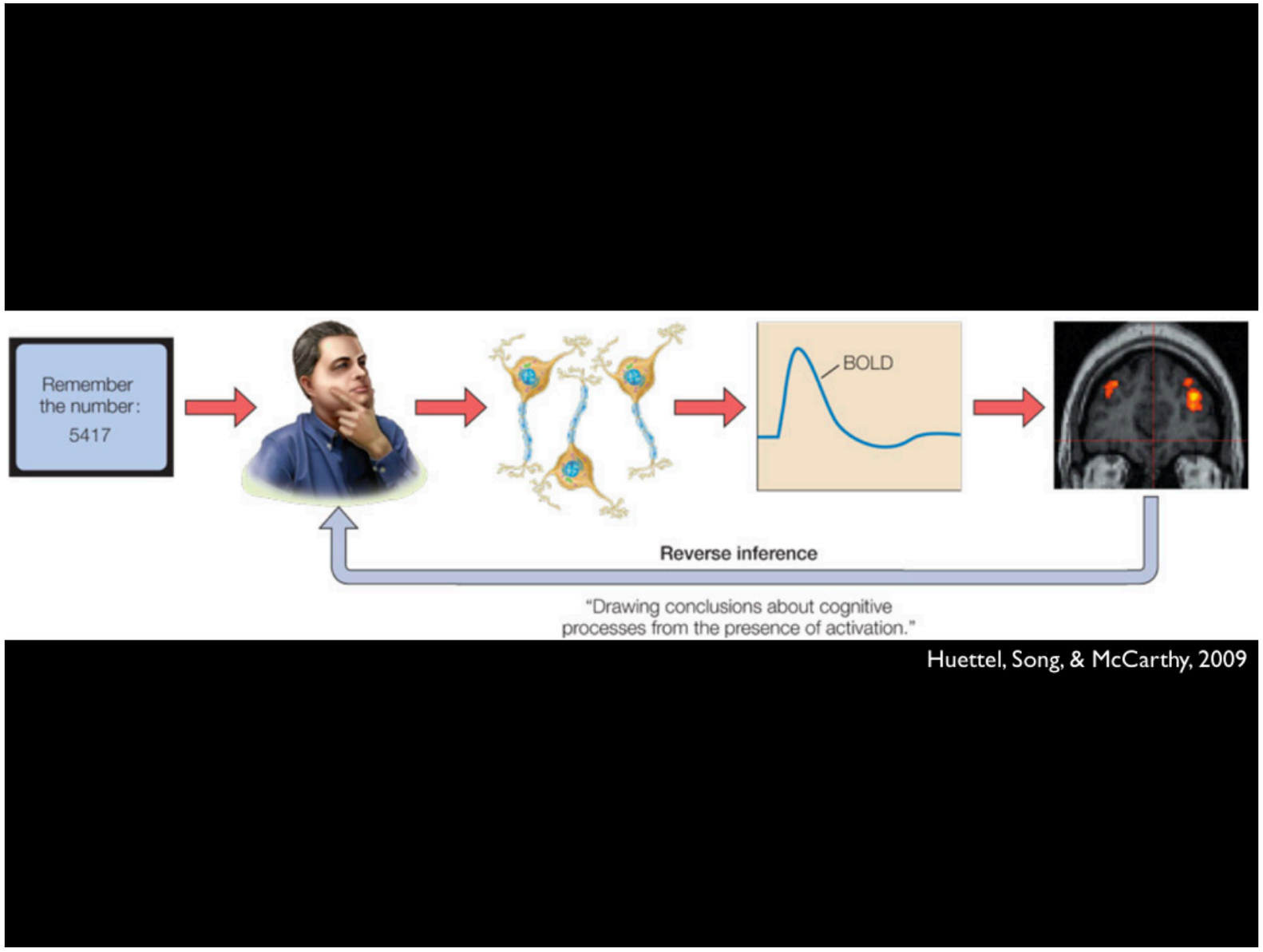


Ziauddeen and colleagues (2012) wrote a review on studies addressing both anticipatory and consummatory neural responding in obese versus lean people. Unfortunately, no clear conclusions could be drawn. Instead they wrote: “The pattern emerging from studies comparing obese individuals and binge-eaters with controls is most remarkable for its variability and inconsistency (p.283).”



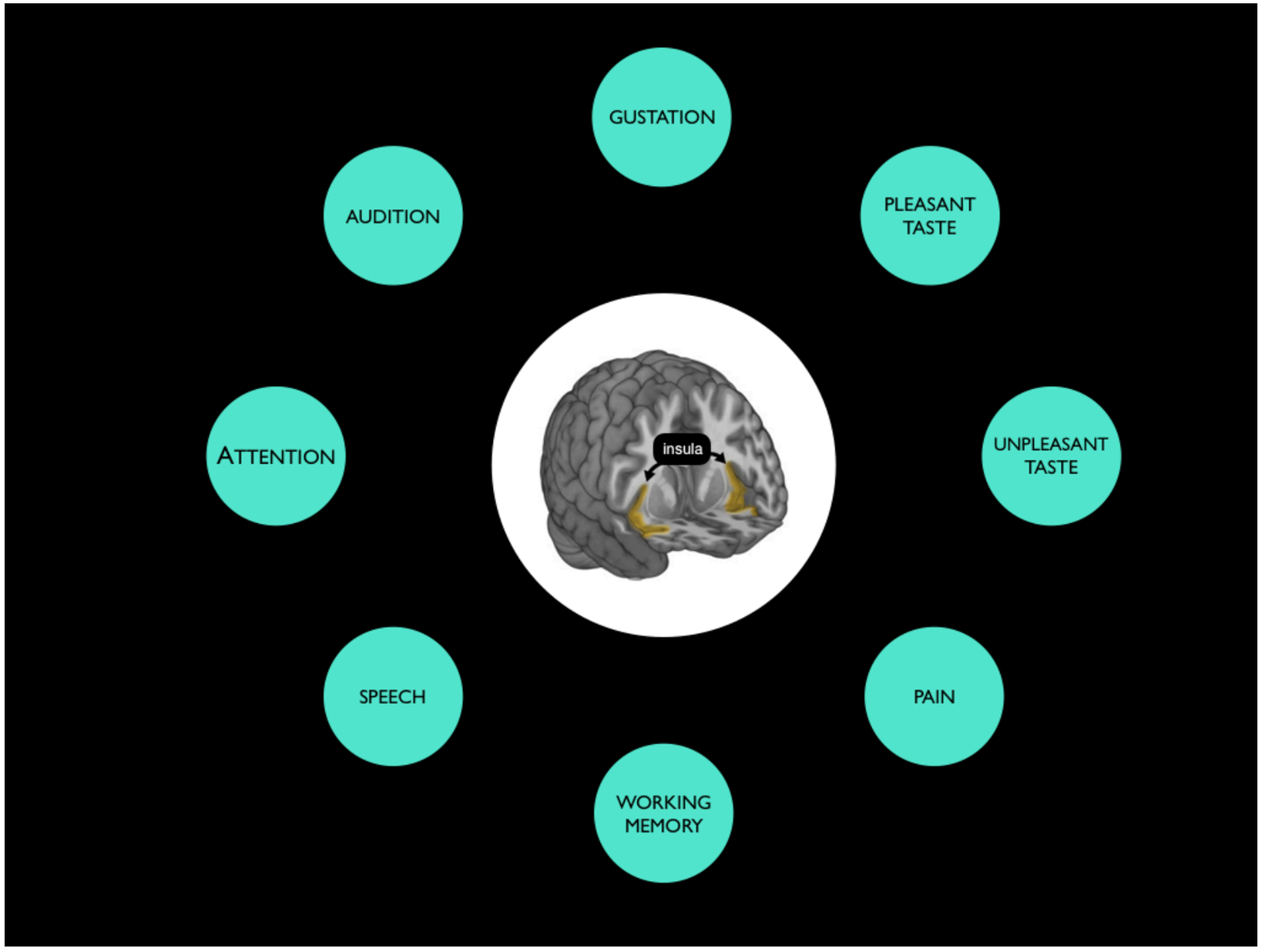
So, though the idea of these imbalances in the reward and control circuits of the brain makes much intuitive sense, empirical support is disappointingly inconsistent. What might be the problem here? Again, the assumption in this line of research seems to be that people firstly and automatically evaluate the hedonic or rewarding aspects of food when presented with food pictures in an MRI scanner.

Just imagine that you lie in the scanner viewing pictures of high-caloric foods. Would you consistently only consider how much you like these foods, or would you think about health, caloric value, your diet, or your body weight as well? Would that maybe even fluctuate during your time in the scanner? So, here as well, I propose that mindset can fluctuate between focusing on hedonic versus health value, within and across participants and studies.

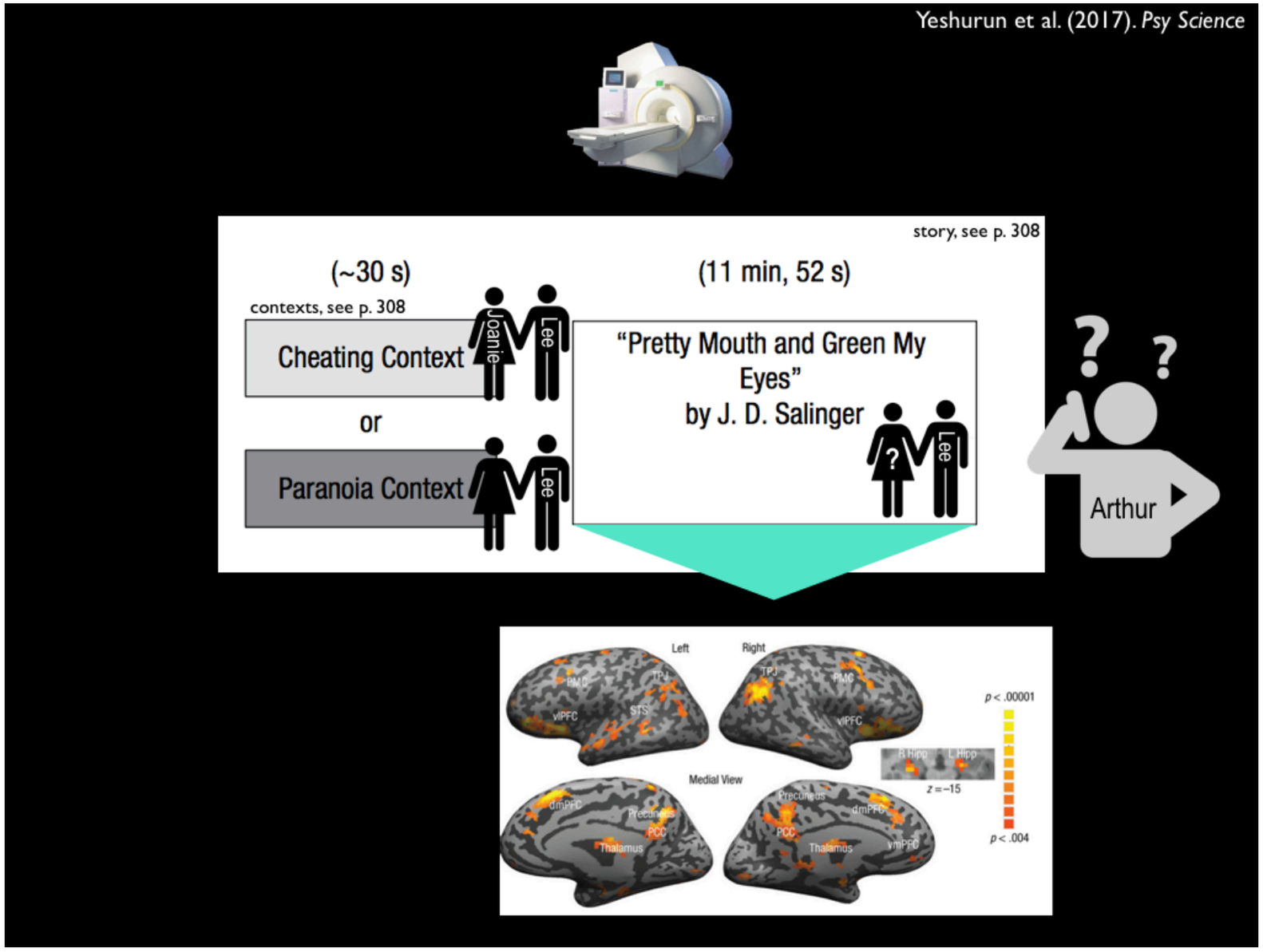


The problem with passive viewing paradigms is that we do not really know what mental process the participant is engaged in. It is important to note here that fMRI is designed to provide information about neural activation that is associated with a known mental process. However, often researchers do the reverse, they deduce a mental state from certain neural activation.

In food-reward research the argument goes like this: "If it is found that the neural response to food stimuli is larger in obese than healthy-weight people in certain areas of the brain that have previously been associated with reward processing, it is concluded that these foods are more rewarding for obese than healthy-weight people" (Roefs et al., 2018, p. 1365). The problem with this line of reasoning is that there is no one-to-one mapping of function to brain area, and that most brain areas are involved in many cognitive functions (Yarkoni et al., 2011).



The insula – frequently reported in studies on food reward – is in fact reported in 1/3 of neuroimaging studies (Yarkoni et al., 2011), and has been associated with very diverse mental functions. If we do not want to get caught in the trap of reverse inference – that is, inferring mental states from neural activity – we need to be pretty sure of the mental process our participant is engaged in. So, my point is that we cannot *assume* that the mental process of hedonic evaluation is going on, we need to be pretty sure. Only then can we draw clear conclusions from our neural findings.

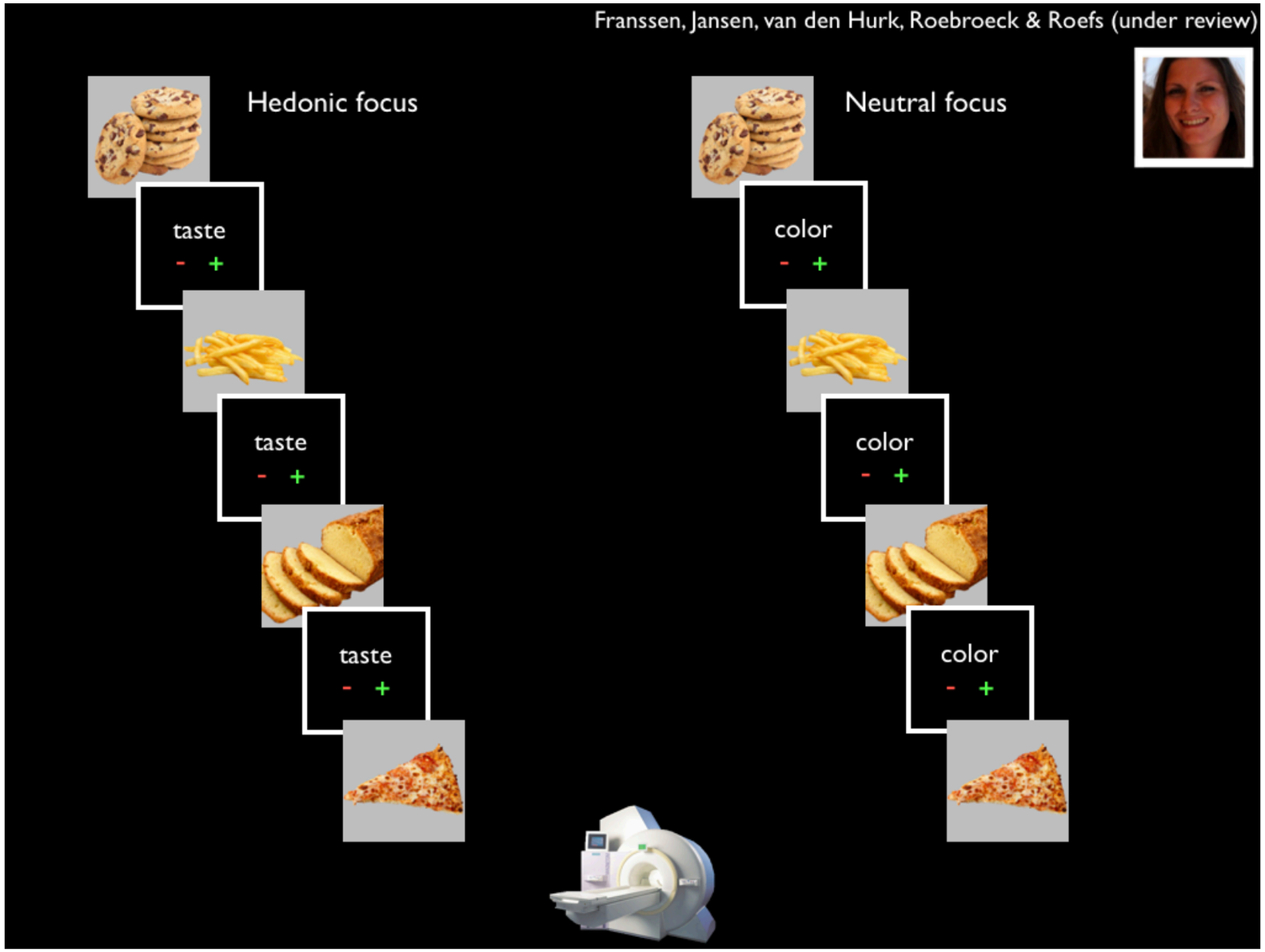


A nice illustration that the brain does not simply respond to presented stimuli, but is affected by prior expectations, by mindset, is a study on neural representations of a story (Yeshurun et al., 2017).

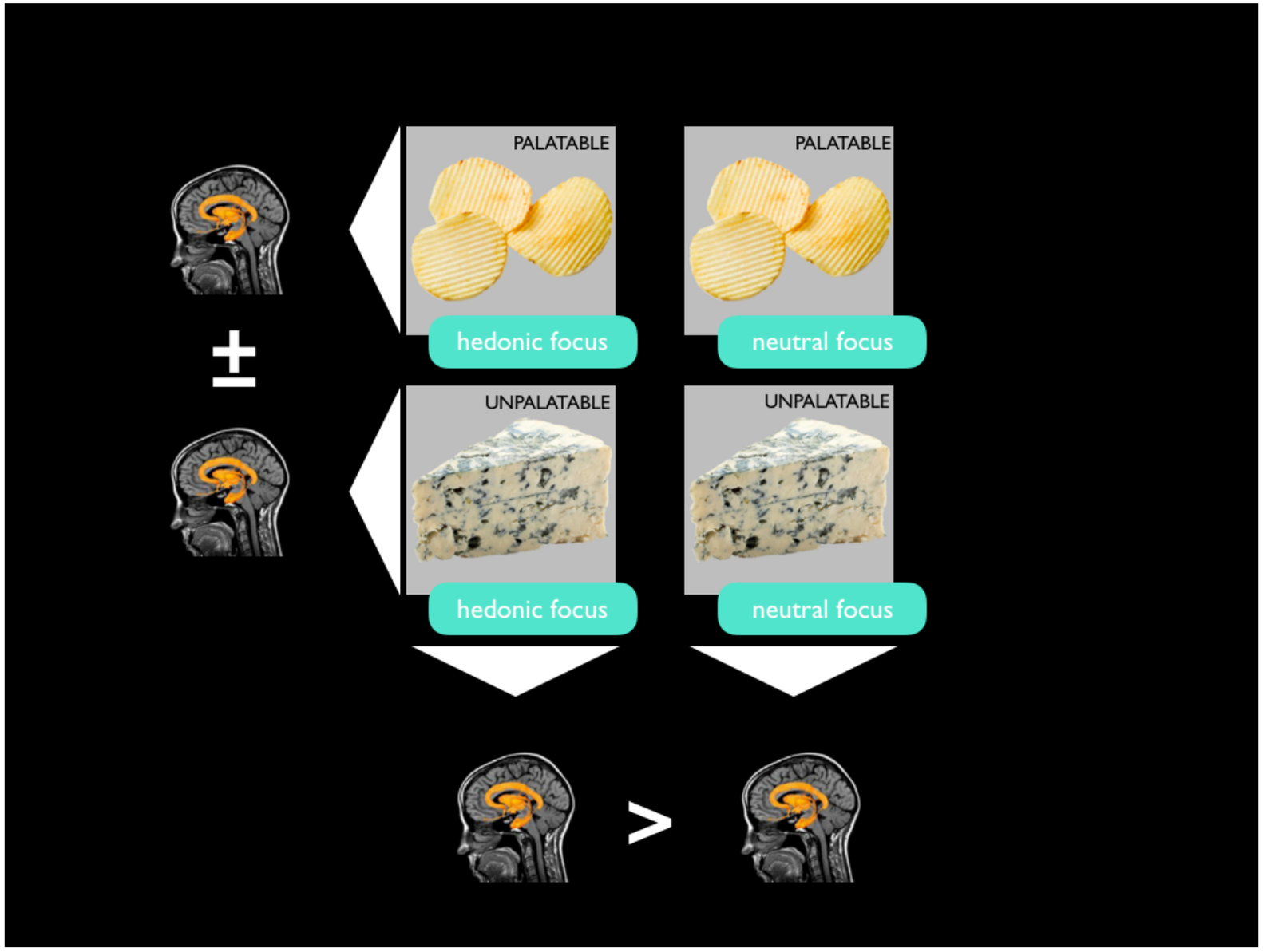
In this study, all participants listened to the same short story while they were in the MRI scanner. "The story was about a phone conversation between two friends, Arthur and Lee. Arthur has returned home after a party after losing track of his wife, Joanie. He is calling Lee to share his concerns over her whereabouts. Lee is at home, and a woman is lying on the bed next to him. The woman's identity is ambiguous—she may or may not be Joanie, Arthur's wife." (p. 308). Crucially, to disambiguate the story, half of the participants were primed with a cheating-context specifying "that Arthur's wife is cheating on him with Lee", whereas the other half were primed with a paranoia-context, specifying that "Arthur is paranoid and that his wife is not cheating on him." (p. 308).

So, keep in mind that neural activity was recorded while all participants listened to the same 11-minute story. The only difference between the two groups was the 30-second priming of context. The researchers found that neural activity was significantly different between the two conditions, while exactly the same story was presented. So, the brain does not simply respond to stimuli that are presented, but neural responding is affected by mindset.

* icon by Akshar Pathak (man on phone) from thenounproject.com.

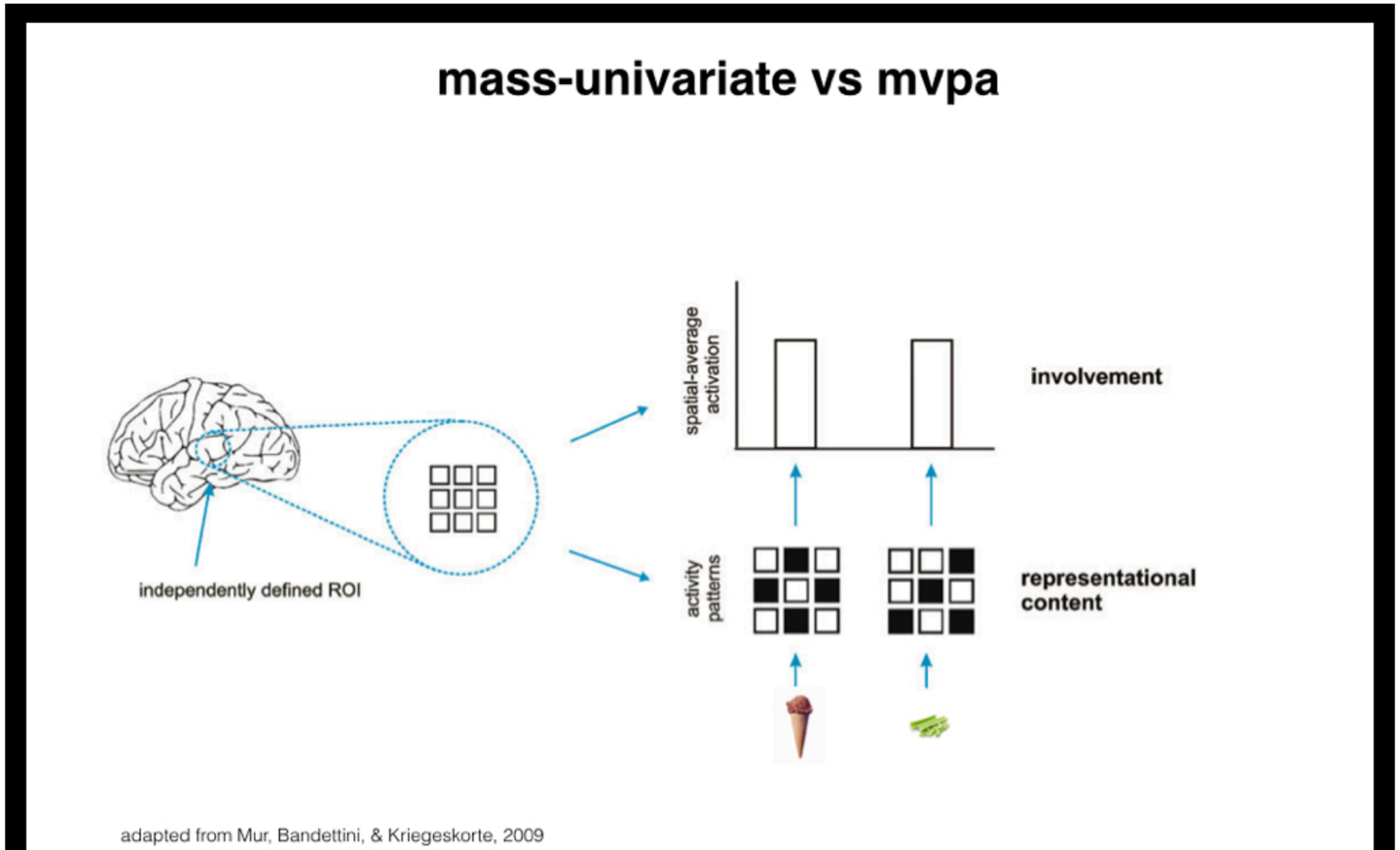


I think the something similar is true for visual food stimuli. In a recent study, with my PhD-student Sieske Franssen, we tried to control the mental process of our participants as much as possible. We either had overweight participants perform a fast-paced hedonic 1-back task (i.e. indicate if the presented food is more or less palatable than the previous one; hedonic attentional focus) or a fast-paced neutral 1-back task (i.e. indicate if the presented food contains more or fewer colors than the previous one; neutral attentional focus).

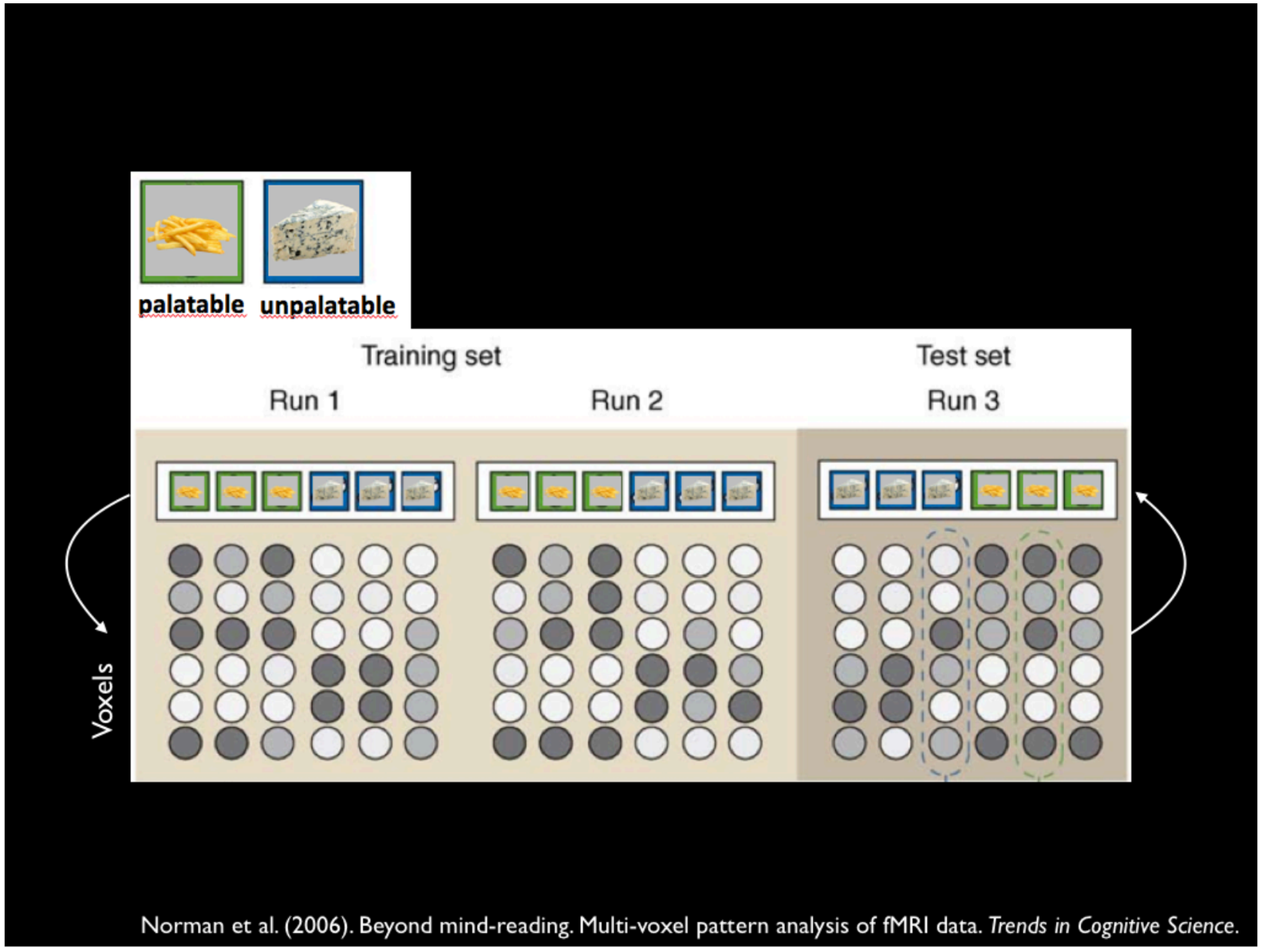


We presented individually tailored highly palatable and highly unpalatable high-caloric food stimuli. These stimuli here represent my preferences, I do like crisps, but really don't like blue cheese. Stimuli were presented with a hedonic focus or a neutral focus. We expected that the reward circuit would respond more strongly to palatable than to unpalatable foods, and specifically with the hedonic focus.

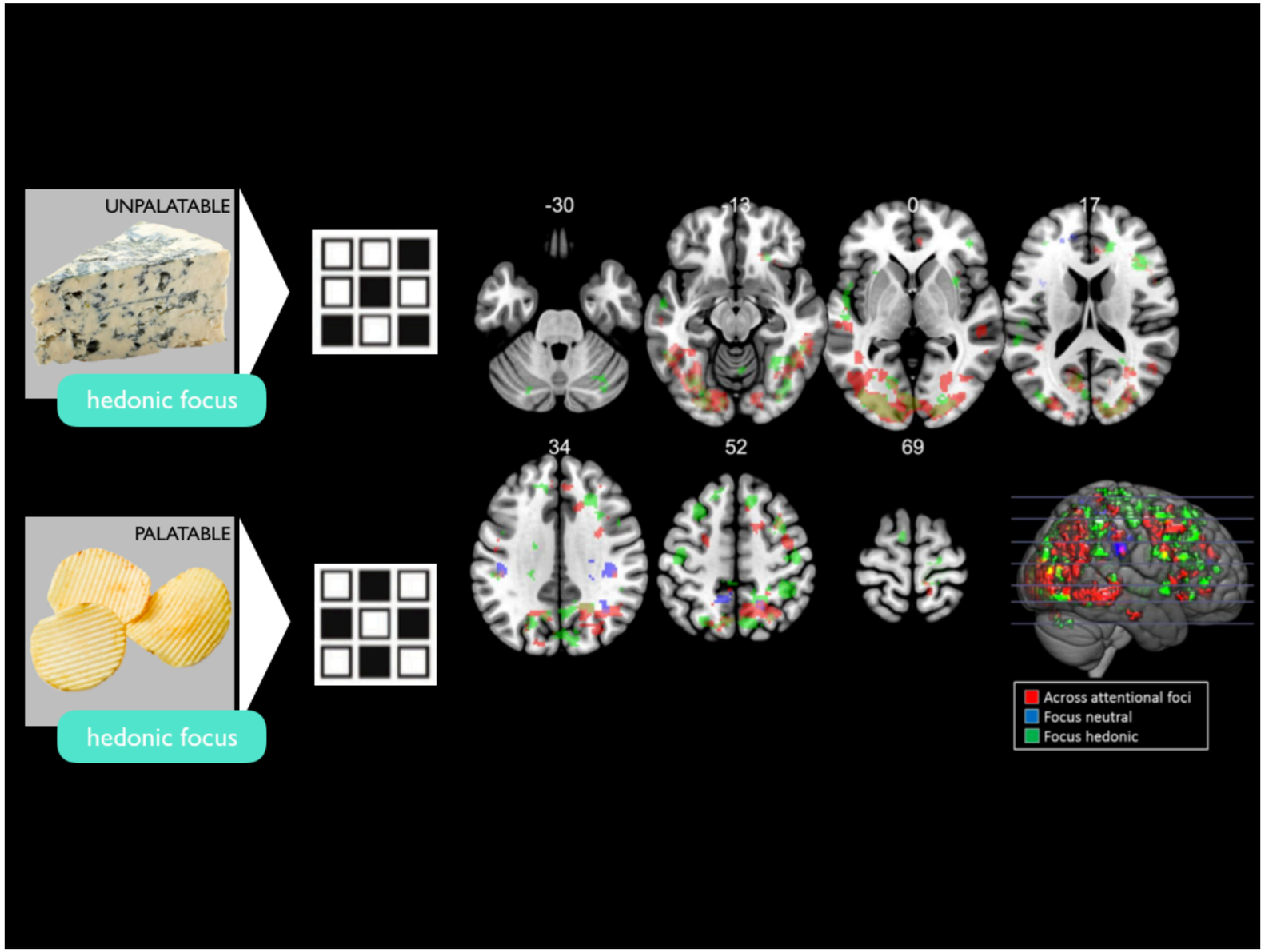
Strikingly, the level of neural activity in the reward-circuit was not significantly different for highly palatable versus highly unpalatable food stimuli, which is what you would expect. Instead, the neural response in several brain regions included in this system was larger with the hedonic attentional focus than with the neutral attentional focus, independent of the palatability of the presented food stimuli. So, neural activity was different between attentional foci while the exact same visual food stimuli were presented. The reward circuitry seems to respond to motivational saliency, which is more pronounced when attention is focused on hedonic evaluation, not food palatability. But that's not the end of the story …



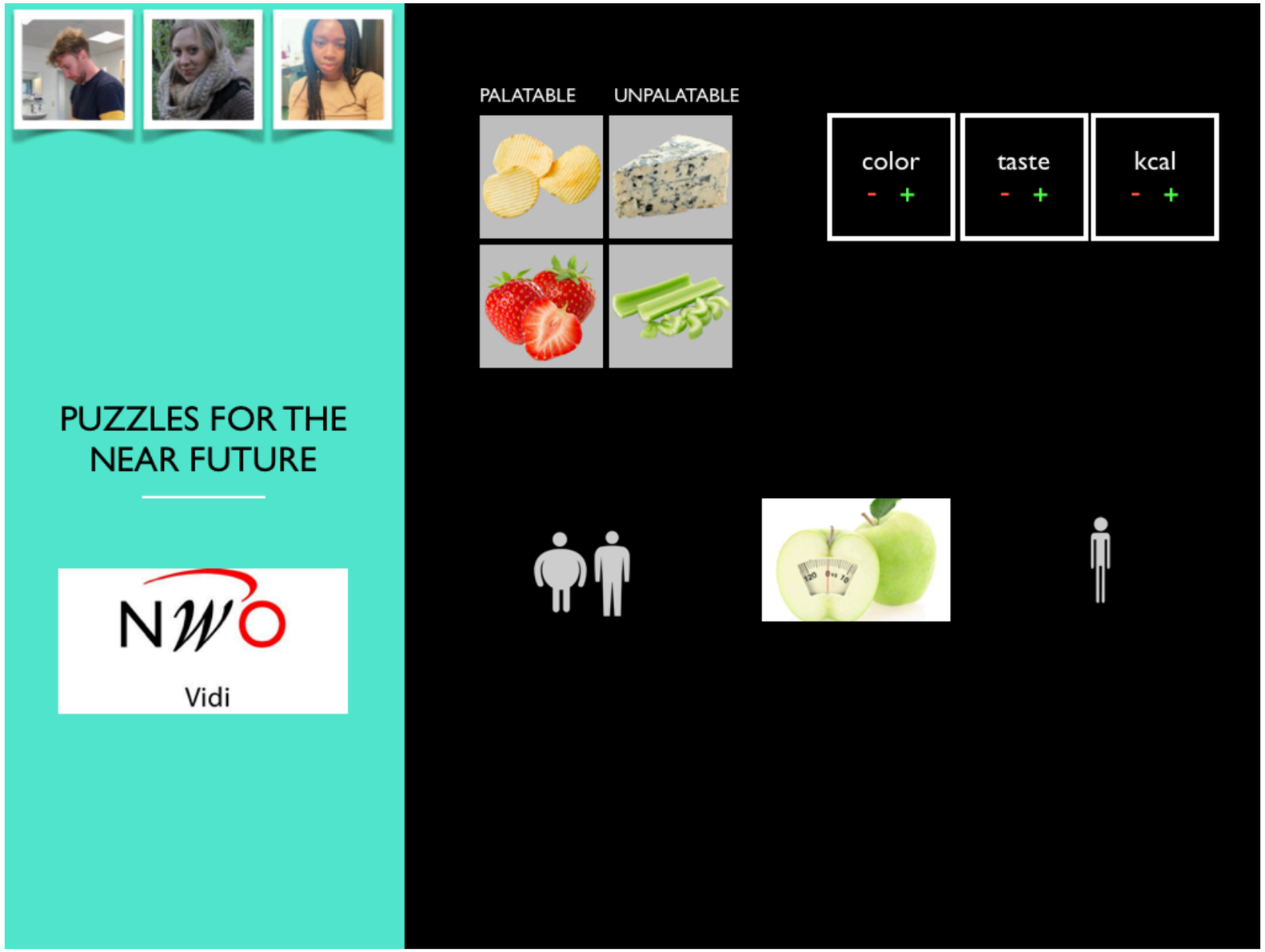
Before I go on, I need to give some more information on fMRI-analysis. On this slide, you can see a hypothetical region of interest of 9 voxels, which are 3D-pixels. In fMRI-analyses, we divide the brain into thousands of voxels. In standard mass-univariate analyses of fMRI data, one looks for regions of neural activation, without consideration of voxel-to-voxel variations in neural activity. These univariate analyses of fMRI data are only informative regarding the *involvement* of certain brain regions in certain tasks. Another type of fMRI-analysis is called multi-voxel pattern analysis (MVPA), in which the researcher looks for multi-voxel patterns within regions of interest or across the whole brain. MVPA of fMRI data decodes *representational content* in the brain (Haxby et al., 2001; Norman, Polyn, Detre, & Haxby, 2006). So, two types of food, for example ice-cream and celery, can *involve* a brain region to a similar degree (result from conventional mass-univariate analyses), but can elicit very different multi-voxel patterns of activity within that brain region (result from MVPA of fMRI data).



Importantly, with MVPA a researcher can test if mental states can be inferred from neuroimaging data (Poldrack, 2011). Training datasets are used to train algorithms what kinds of neural patterns relate to what kind of mental states (here, viewing palatable versus unpalatable foods). Next, the accuracy with which the algorithm can predict the mental state from the pattern of neural activity is tested in a separate dataset. So, with MVPA a researcher can estimate to what extent a pattern of brain activation is predictive of a certain mental process. Thereby, MVPA provides a formal method of reverse inference (Poldrack, 2011).



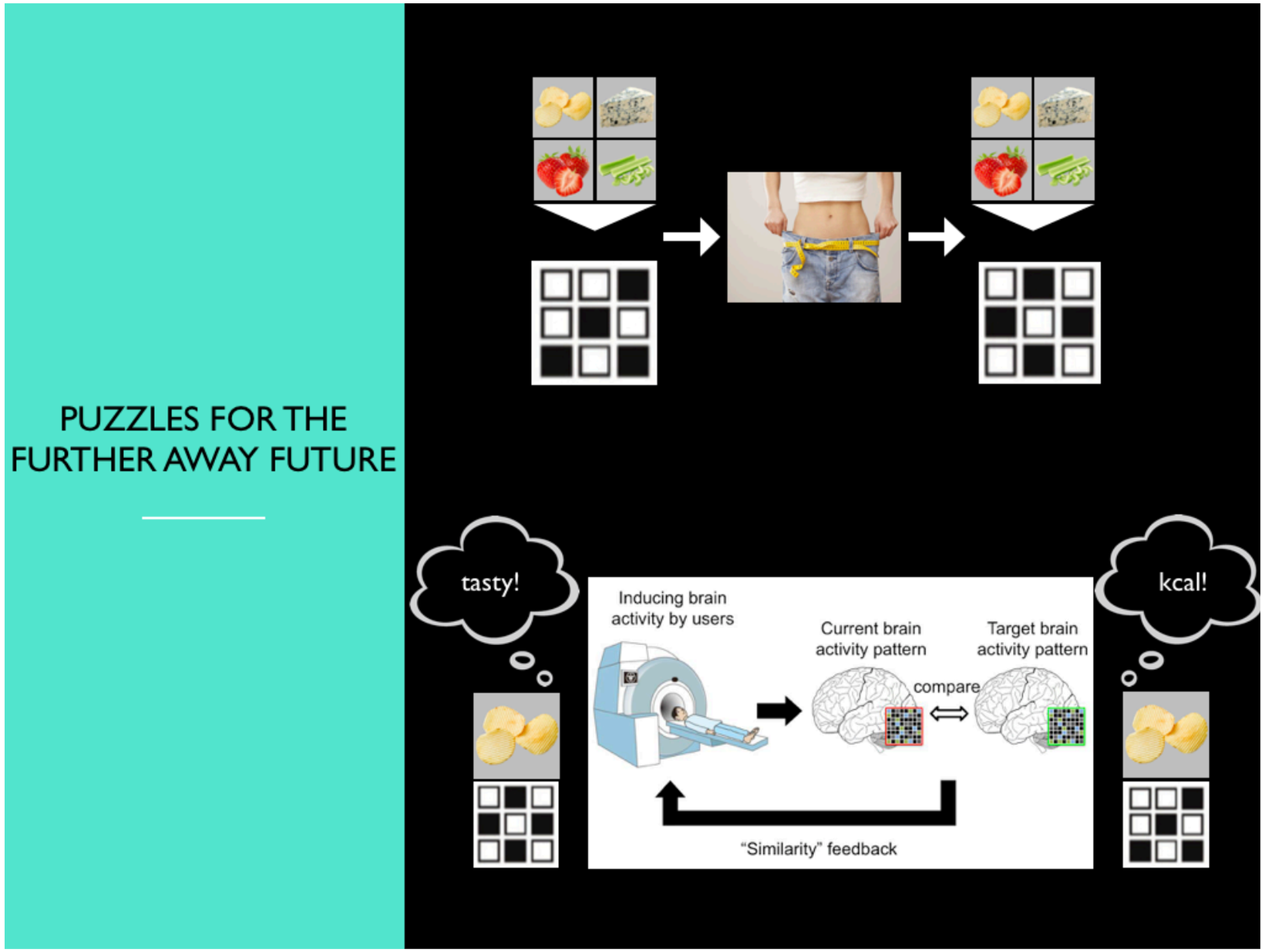
For the study I just presented, we also performed MVPA, and we showed that food palatability could be specifically decoded from multivoxel patterns of neural activity. So multi-voxel patterns of palatable foods were significantly different from those of unpalatable foods. Moreover, this decoding of palatability was mostly exclusively successful for the hedonic focus, not for the neutral focus. So, the representational content of the brain reflects the attentional focus during task performance. How you look at food, affects how your brain represents it.



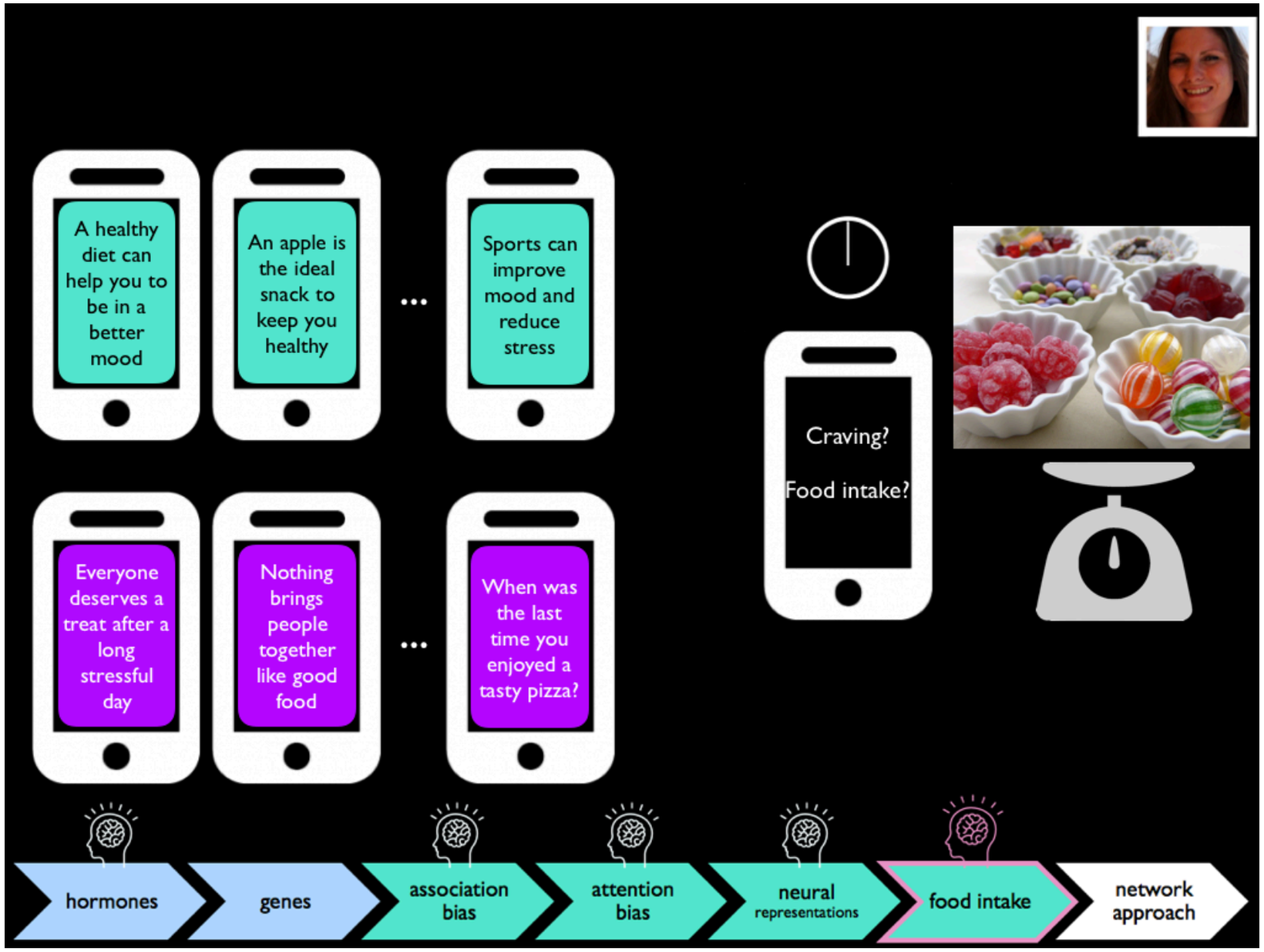
In my ongoing VIDI project, we are building on this work, and we try to solve some more puzzles. For our fMRI-work, we added low-caloric food stimuli, and we added a condition in which we ask people to evaluate caloric density. We aim to find out how caloric density is neurally represented, and if neural representations of food only reflect caloric density if participants are actively evaluating it, like with the palatability in our previous study. Moreover, we study how these neural representations are moderated by BMI and levels of chronic dietary restraint. In a related project, we will study how this works in anorexia nervosa patients.

* icon by Daniel Behrends (male icon) from thenounproject.com.

PUZZLES FOR THE FURTHER AWAY FUTURE

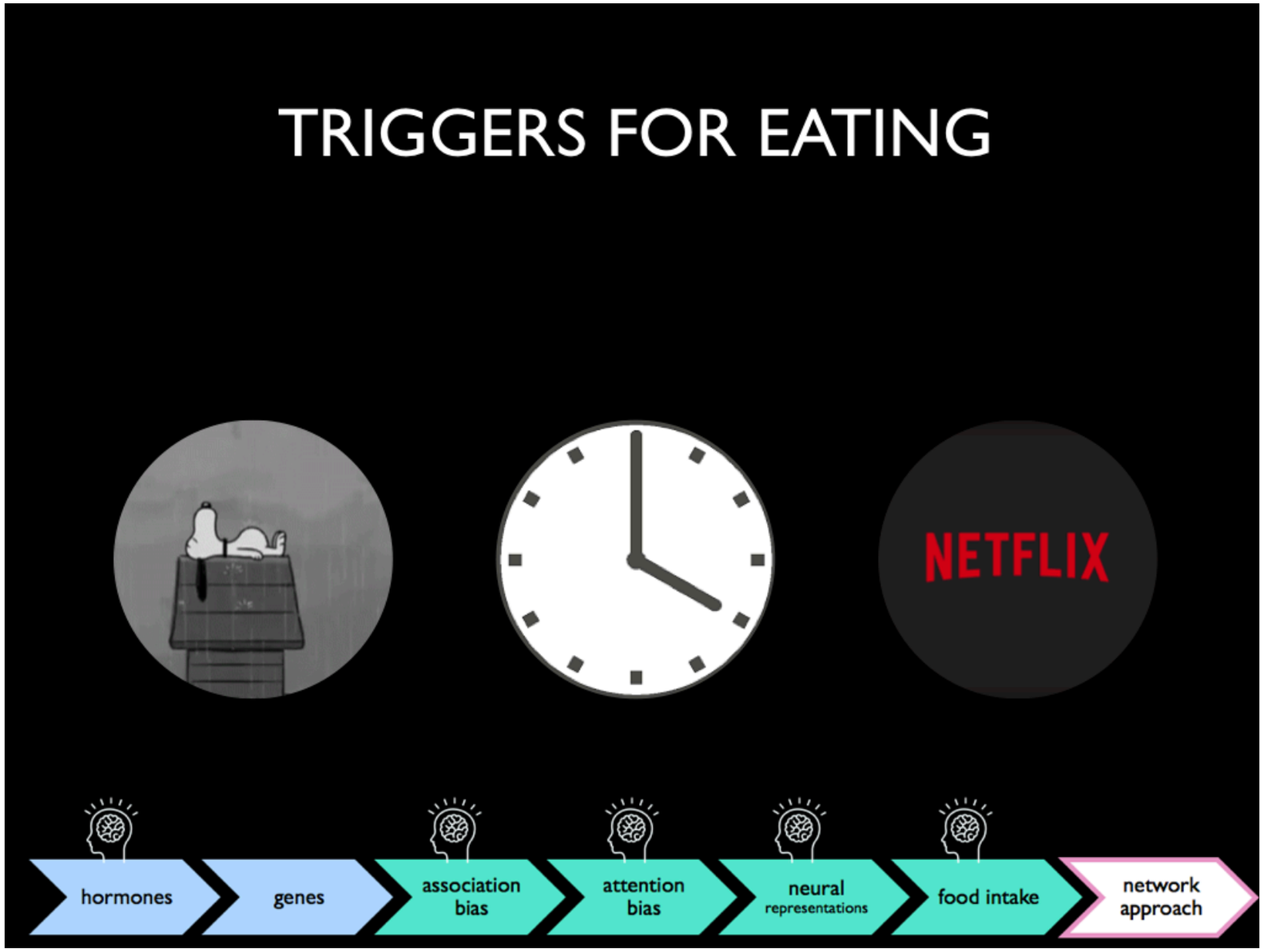


In future neuroimaging research, I would be interested in studying how a dietary intervention may change these neural representations of food, and if the degree of weight loss achieved by a diet correlates with changes in neural representations of food. Also, I would like to examine whether decoded neurofeedback would be a valuable approach of training these neural representations of food – for example, making them reflect health-value – and thereby influence behavior.

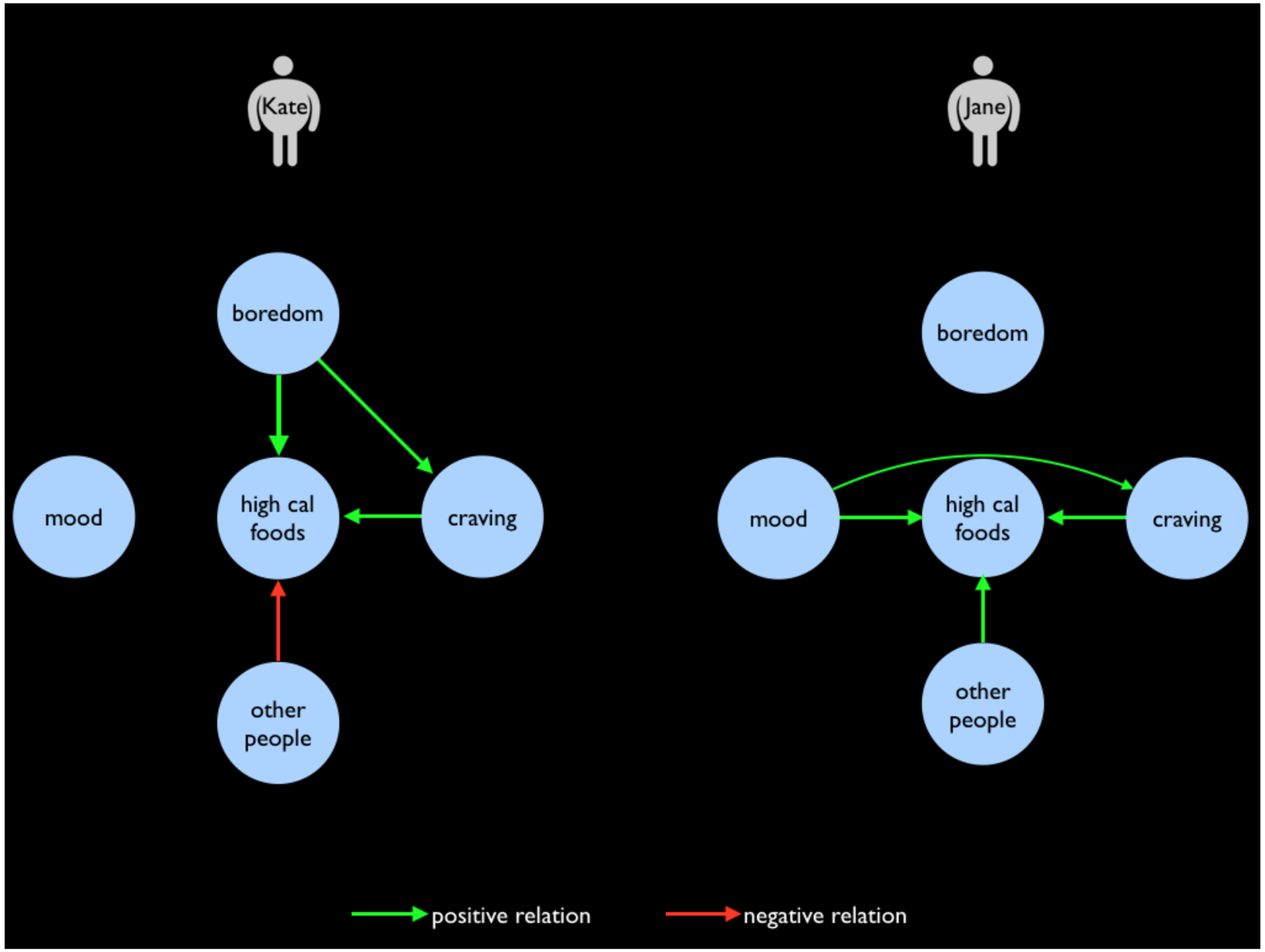


If we can indeed show that neural representations of food are so flexible, depending on people's current attentional focus, it may suggest that the attentional focus, the mindset, is a good target for intervention. An intervention should aim to increase the time that overweight people are in a health-mindset. We are currently running a change-your-mindset study, in which we send either hedonic mindset or health mindset inspirational messages to the participant's smartphone multiple times a day. We measure food craving and food intake both in real life with an app and in the lab.

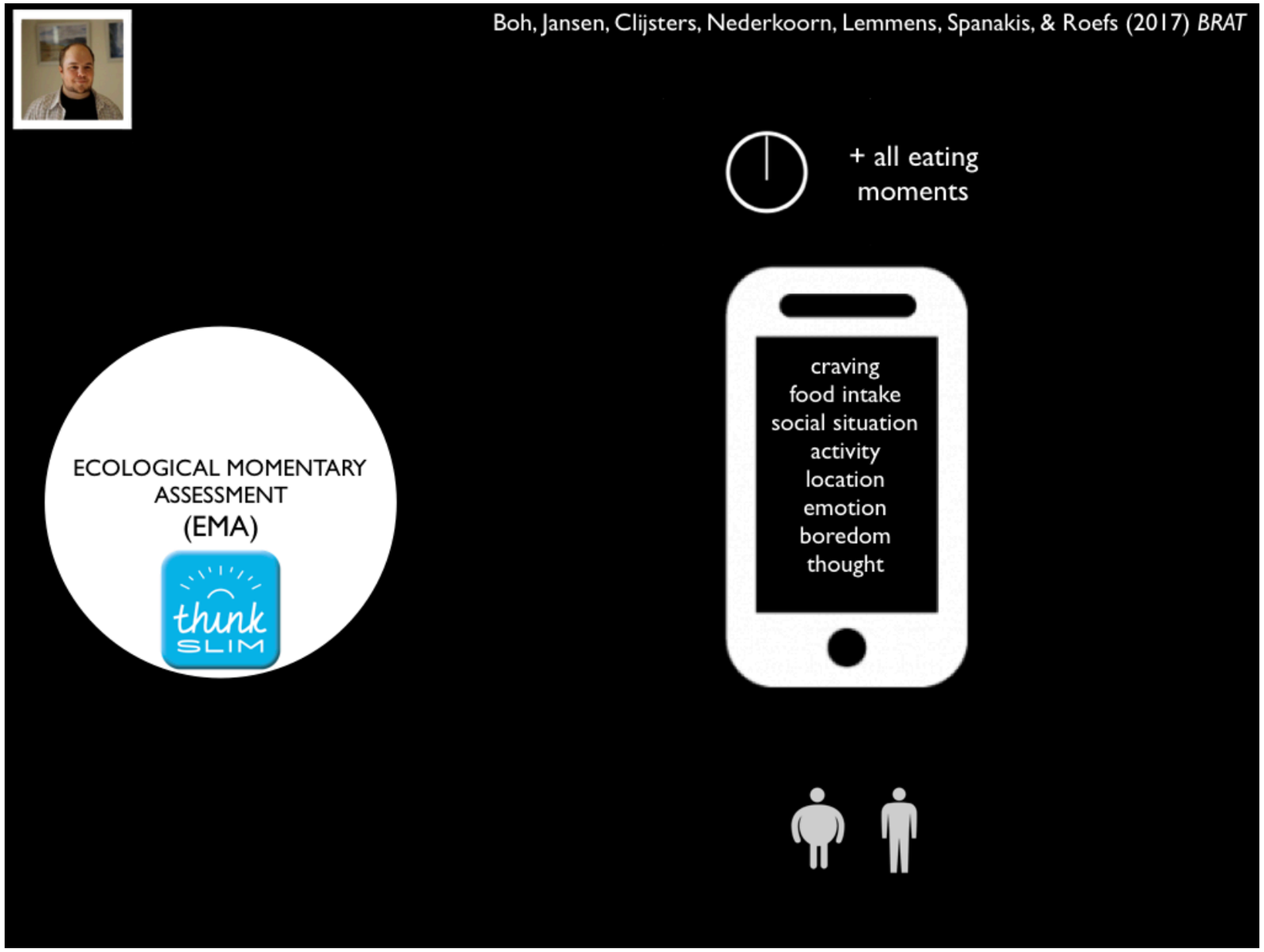
This approach is in the spirit of cognitive behavioral therapy, CBT, which aims to change the way people think. CBT is currently the most successful therapy for eating disorders, and research from our group has shown that CBT holds promise for obesity as well, and can prevent relapse (Werrij et al., 2009).



Taken together, I think I have established that food cues are not always enticing, but can actually also remind you of your diet, depending on your current mindset. Taking it one step further, not only food cues can lead to food consumption, but also other cues. For example, a negative mood, a certain time of day, watching your favorite Netflix series, etc. Which cues are associated with food intake likely varies across people and also within a person across time.



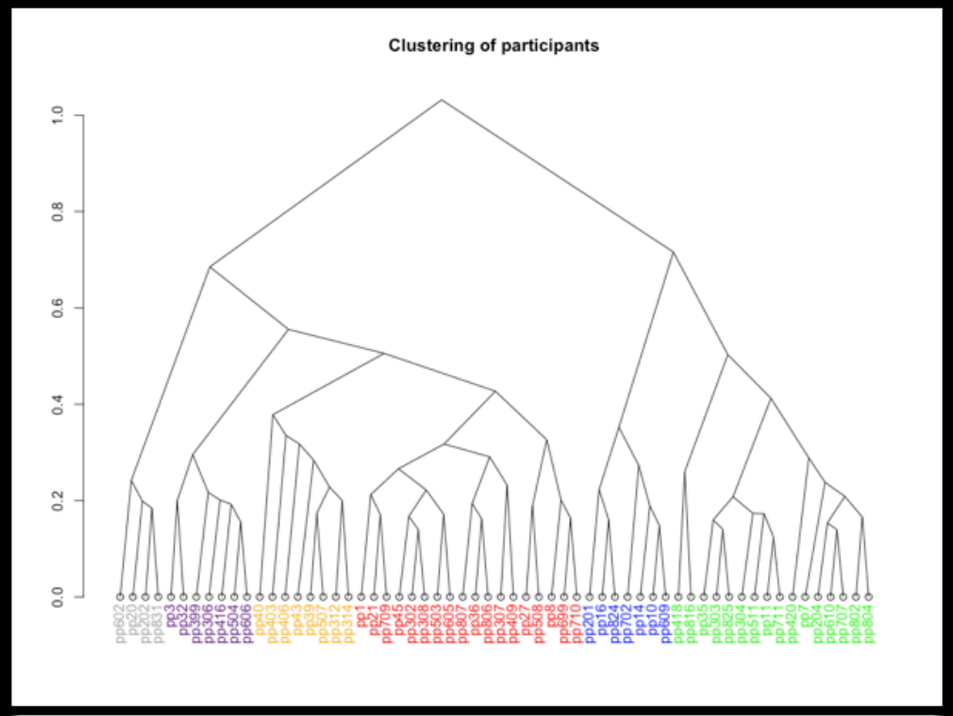
Meet for example Kate and Jane, they are both overweight, but they have quite different triggers for their food intake. As you can see in this network of variables, with each variable represented by a node, for Kate the intake of high-caloric foods is mostly triggered by boredom and when she is alone. For Jane, food intake is mostly triggered by a happy mood, and when she has company. So, Kate and Jane are at risk of consuming high caloric foods in quite different situations, and may require intervention at different times.



For constructing these individual networks, it is necessary to measure relevant variables multiple times a day within each person. In our project, Think Slim, we used ecological momentary assessment (EMA) to obtain measures of craving, food intake, social situation, activity, location, emotion, boredom, and the thought participants had when about to eat something. EMA is implemented on people's smartphones and has a high ecological validity. We collected these data in overweight and healthy-weight people (Boh et al., 2017; Spanakis, Weiss, Boh, & Roefs, 2016).

* icon by Daniel Behrends (male icon) from thenounproject.com.

Spanakis, Weiss, Boh, Kerkhofs, & Roefs (2016). *Procedia Computer Science*



evening at home eaters

outdoor social eaters

circumstances driven eaters

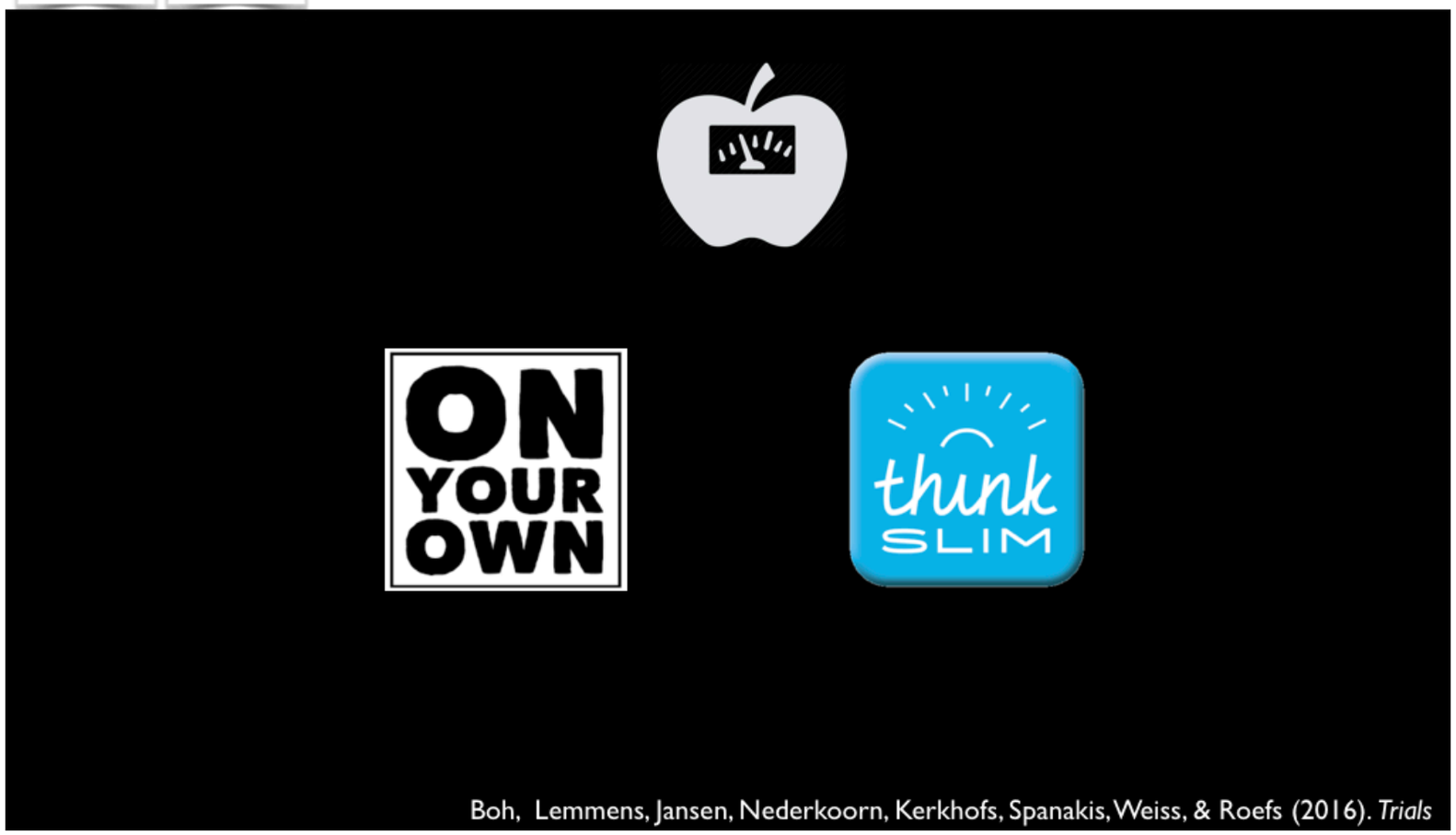
very occasional eaters

after activity snackers

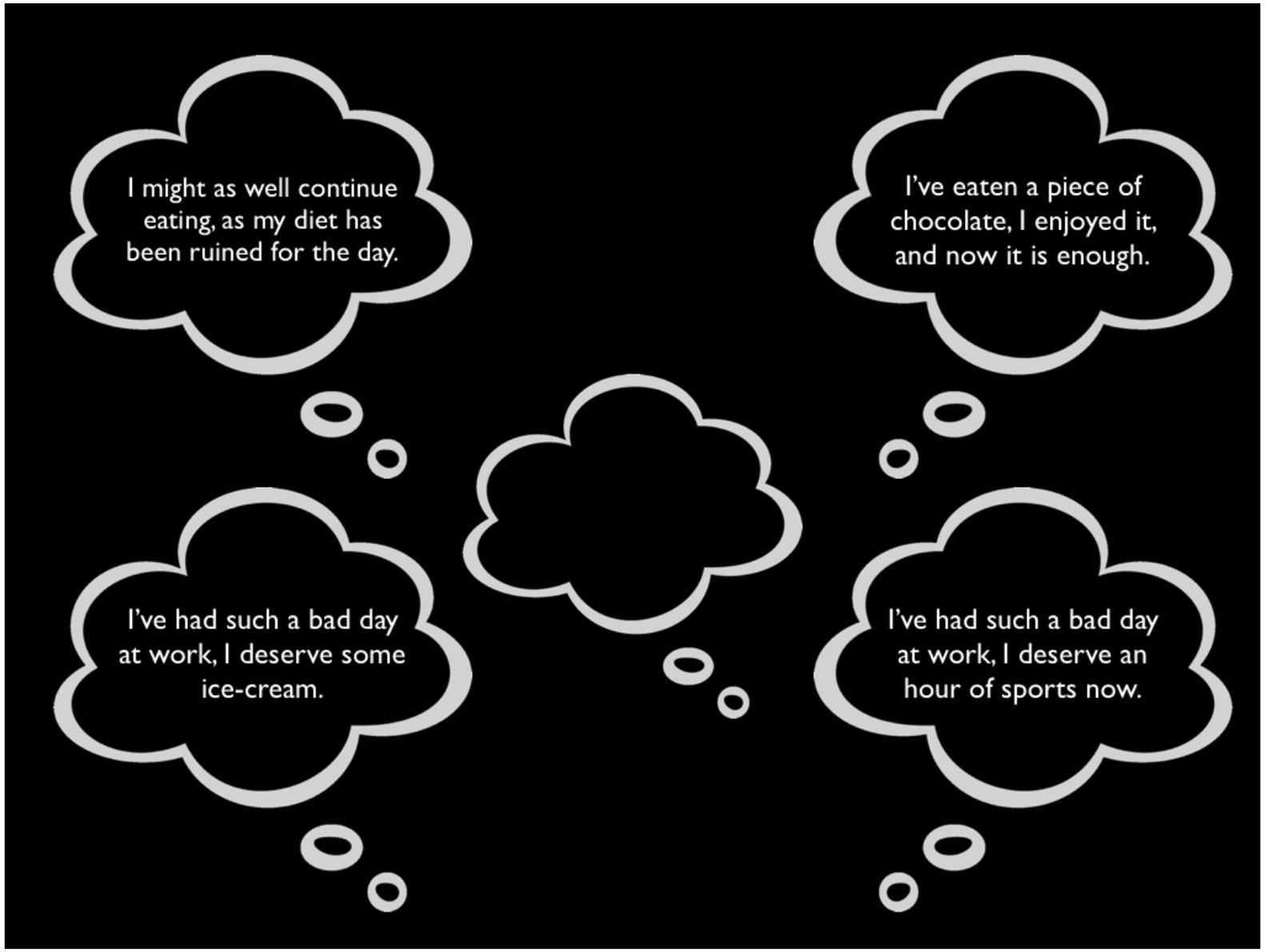
unhealthy craving satisfaction

We used the data of the overweight people to arrive at different profiles of eating behavior, by using a clustering algorithm. This resulted in six different types of eaters. One of these types could for example be characterized as ‘evening-at home eaters’ and another type as ‘after-activity snackers’ (Spanakis, Weiss, Boh, Kerkhofs, & Roefs, 2016).

THINK SLIM

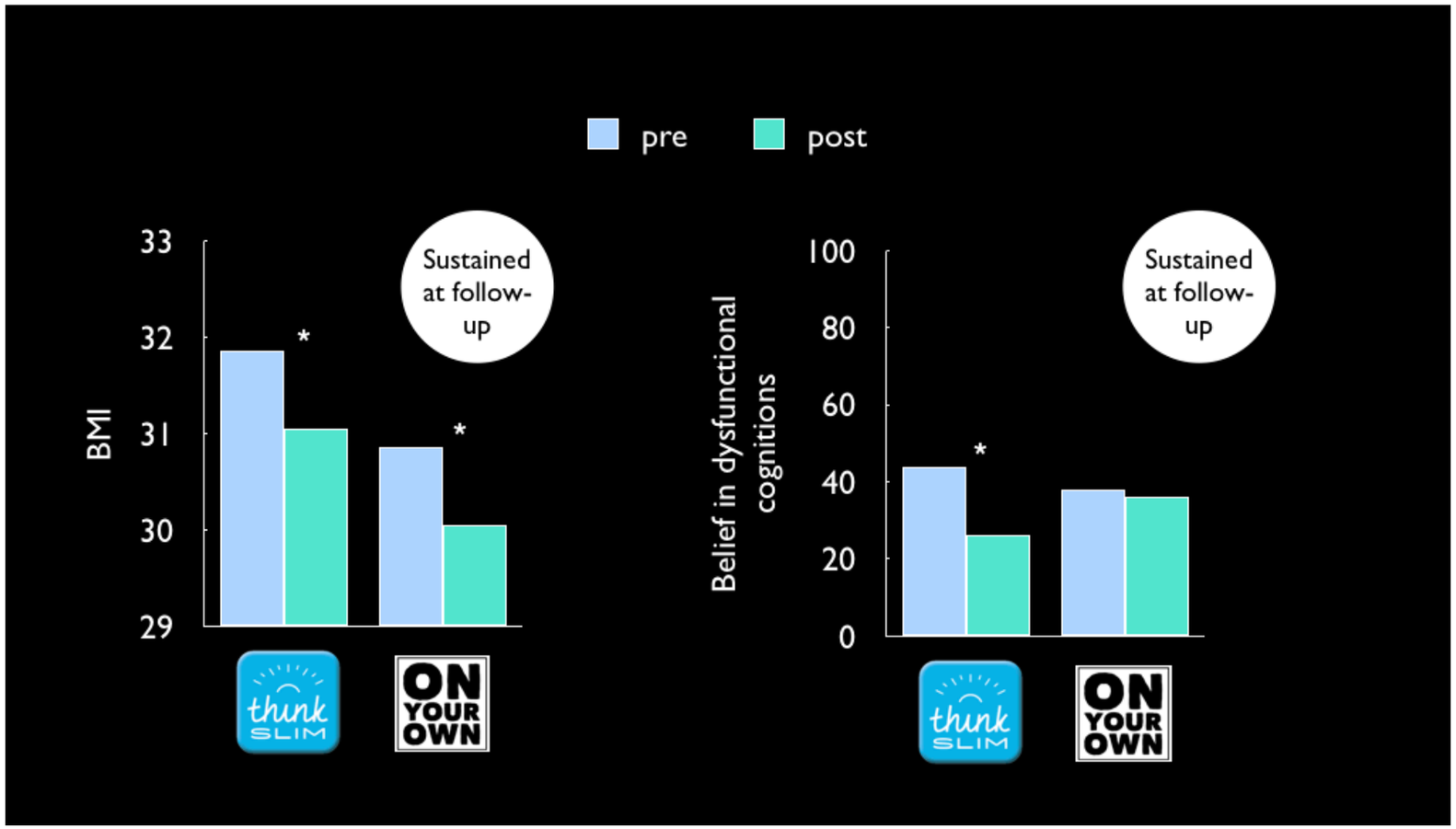


In a second study of Think Slim, we based individualized Cognitive Behavioral Therapy, CBT, on these profiles and compared our Think Slim condition to a ‘dieting on your own condition’ (Boh et al., 2016).



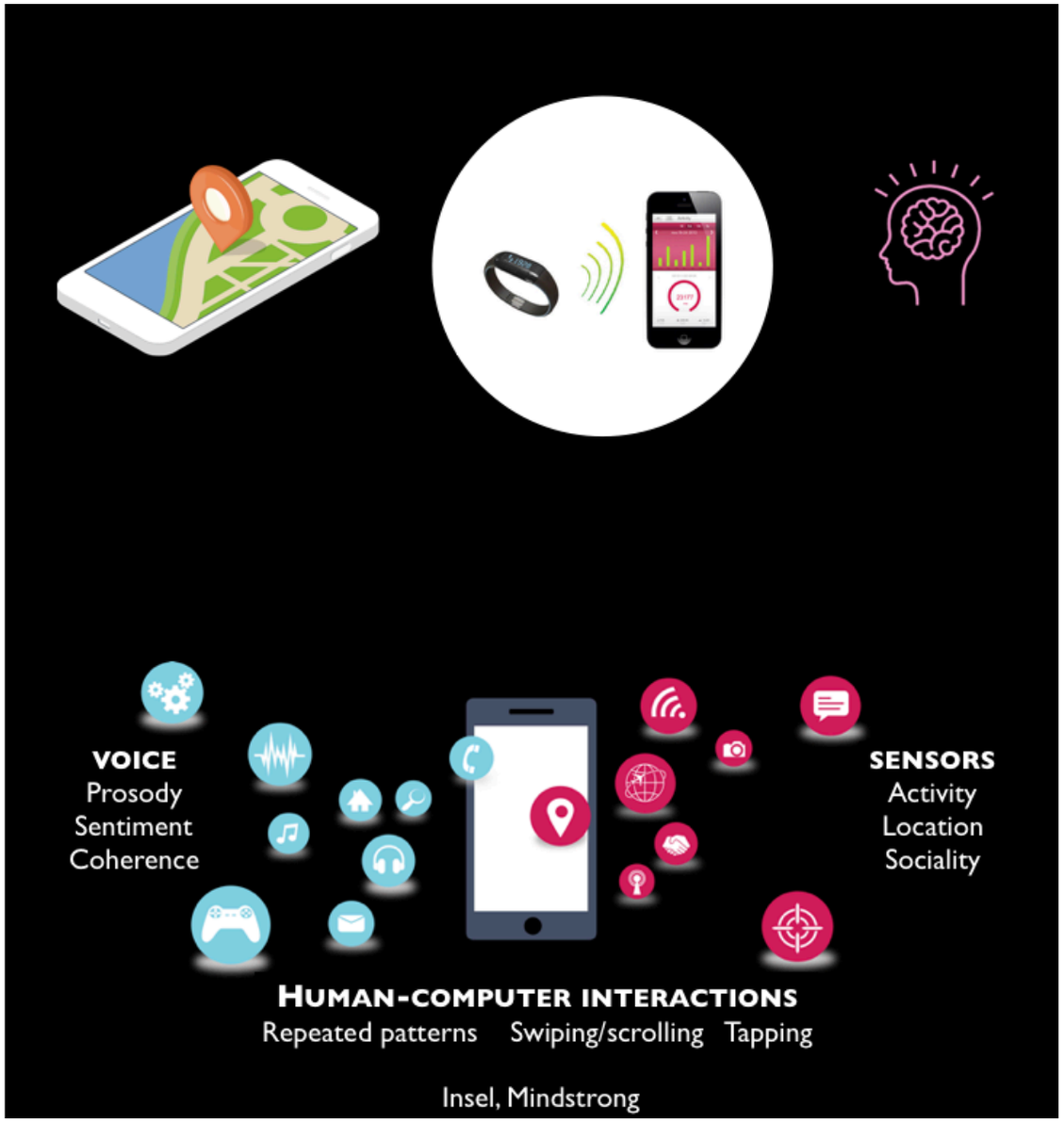
CBT is aimed at challenging so-called dysfunctional thoughts, thoughts that undermine dieting attempts. Some of these thoughts may be familiar, such as "I've already eaten this chocolate, so I might as well continue eating, as my diet has been ruined for the day", or "I have had such a bad day at work, I deserve some ice-cream". In CBT, participants learn – via all kinds of techniques – to transform these diet-undermining thoughts into diet-supporting thoughts, such as "I've eaten a piece of chocolate, I enjoyed it, and now it is enough", or "I've had such a bad day at work, I deserve an hour of sports now.".

PRELIMINARY RESULTS
THINK SLIM



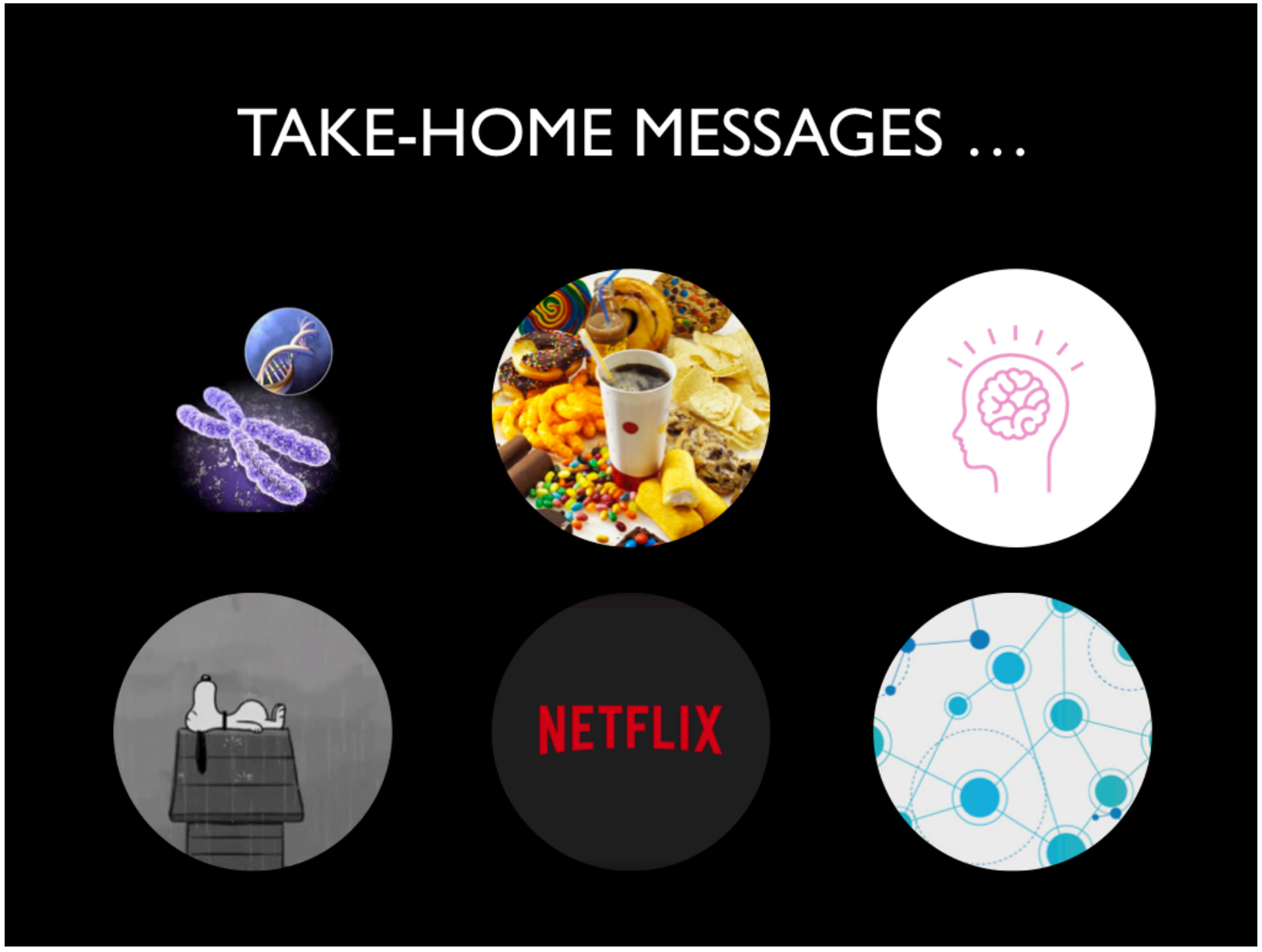
Our preliminary analyses showed that participants lost about 0.8 BMI points in 6 weeks in both conditions, and this weight loss was maintained at follow-up. At the time of follow-up all participants had received Think Slim, due to ethical reasons. Reductions in dysfunctional cognitions related to eating and self-reported dysfunctional eating were specifically only occurring in the Think Slim condition, which makes sense as Think Slim was targeting these variables. Interestingly, people who regained weight at follow-up, scored higher on these measures of dysfunctional cognitions and eating at follow-up.

FUTURE



In future research, I would like to further build on this method and these results. In Think Slim we fully relied on self-reported data, but in the future, I would like to add sensor-data, such as geolocation and physical activity. Also, I think it is important to optimize the sampling of data and the prediction of high-caloric eating moments, and thereby provide more timely and better-informed warnings. Moreover, these warnings may be focused on inspiring a healthy attentional focus.

Finally, an interesting research program, called digital phenotyping has recently started to develop. Digital phenotyping uses the smartphone as a tool for objective and ecologically valid measurements. This method relies on sensor technology, characteristics of voice and speech, and on human-computer interaction (Insel, 2018). Imagine for example that by this method you discover a pattern over several weeks that a person takes long to respond to messages, is browsing online until late at night, and is mostly at home. You may then suspect that it is not going particularly well with this person, and your suspicion may be increased by the tone, timing, and content of this person's social media posts. I would be interested in studying how digital phenotyping could be useful for understanding obesity, for predicting high caloric food intake. This is not only a research challenge, but of course also an ethical challenge.



I am almost at the end of my lecture now, and it is time for some take-home messages. I hope I have convinced you that a risky genetic profile or the obesogenic environment are by themselves not sufficient explanations for the obesity epidemic. Our obesogenic environment does not only *not* have the same effect on everyone, the same person is not always affected similarly. There is an interaction with the person's current mindset, which on its turn may be affected by for example physiological or emotional state. Moreover, what makes our environment obesogenic, is not the same for everyone. There can be different triggers in our society that lead to the consumption of high-caloric foods for different people.

So, everything should be kept as simple as possible, but not simpler. Your mind may be your biggest asset in beating the power of food, by adopting a healthy attentional focus or mindset. This attentional focus or mindset affects your hunger-hormone level, how much you attend to food, your associations with food, your neural representations of food, and … how much you eat.

Until now, obesity has been studied and treated mostly from a biomedical perspective, but as obesity is in essence a regulatory problem, it needs more attention from psychological science. To achieve sustainable weight loss, a long-term lifestyle change is necessary. A doctor advising people to do that, or just prescribing a diet, will not suffice, certainly not in the long run. Future research should aim to elucidate cognitive and neural mechanisms underlying regulation, to more effectively help people achieve the necessary long-term changes. That - in my mind - is real lifestyle medicine.

References

- Boh B, [...] & Roefs A (2016). Indulgent thinking? Ecological momentary assessment of overweight and healthy-weight participants' cognitions and emotions. *Behaviour Research and Therapy, 87*, 196-206.
- Boh B, [...] & Roefs A (2016). An ecological momentary intervention via smartphone and internet: Study protocol for a randomized controlled trial for weight loss and healthy eating. *Trials*, 17:154.
- Castellanos EH et al. (2009). Obese adults have visual attention bias for food cue images: evidence for altered reward system function. *International Journal of Obesity, 33*, 1063-1073.
- Crum AJ, Corbin WR, Brownell KD & Salovey P (2011). Mind over milkshakes: Mindsets, not just nutrients, determine ghrelin response. *Health Psychology, 30*, 424-429.
- Davids S et al. (2010). Increased dorsolateral prefrontal cortex activation in obese children during observation of food stimuli. *International Journal of Obesity, 34*, 94-104.
- Field M, Werthmann J, Franken I, Hofmann W, Hogarth L & Roefs A (2016). The role of attentional bias in obesity and addiction. *Health Psychology, 35*, 767-780.
- Franssen S, Jansen A, van den Hurk J, Roebroeck A & Roefs A (under review). Power of mind: attentional focus rather than palatability dominates neural responding to visual food stimuli.
- Hofmann W, Friese M, Strack F (2009). Impulse and self-control from a dual-systems perspective. *Perspectives on Psychological Science, 4*, 162-176.
- Haxby JV et al. (2001). Distributed and overlapping representations of faces and objects in ventral temporal cortex. *Science, 293*, 2425-2430.
- Insel TR (2018). Digital phenotyping: a global tool for psychiatry. *World Psychiatry, 17*, 276-277.
- Kamperman M (26-2-2019). Column in Volkskrant: https://www.volkskrant.nl/wetenschap/ik-ben-hoogleraar-maar-weet-nauwelijks-hoe-het-zit~b23e202f/?referer=https%3A%2F%2Fwww.google.com%2F
- Khera AV et al. (2019). Poloygenic prediction of weight and obesity trajectories from birth to adulthood. *Cell, 177*, 587-596.
- Konttinen H et al. (2015). Appetitive traits as behavioural pathways in genetic susceptibility to obesity: a population-based cross-sectional study. *Scientific Reports*, 5:14726.
- Lean MEJ & Malkova D (2016). Altered gut and adipose tissue hormones in overweight and obese individuals: cause or consequence. *International Journal of Obesity, 40*, 622-632.
- Liu Y, Roefs A, Werthmann J & Nederkoorn C (2019). Dynamics of attentional bias for food in adults, children, and restrained eaters. *Appetite*, *135*, 86-92.
- Llewellyn CH & Fildes A (2017). Behavioural susceptibility theory: Professor Jane Wardle and the role of appetite in genetic risk of obesity. *Current Obesity Reports, 6*, 38-45.
- Llewellyn CH & Wardle J (2015). Behavioral susceptibility to obesity: Gene-environmnet internal in the development of weight. *Physiology & Behaviour, 152*, 494-501.
- Loeber S et al. (2012). Impairment of inhibitory control in response to food-associated cues and attentional bias of obese participants and normal-weight controls. *International Journal of Obesity, 36*, 1334-1339.
- Mur M, Bandettini PA & Kriegeskorte N (2009). Revealing representational content with pattern-information fMRI - an introductory guide. *Social Cognitive and Affective Neuroscience, 4*, 101-109.
- Norman KA, Polyn SM, Detre GJ & Haxby JV (2006). Beyond mind-reading: multi-voxel pattern analysis of fMRI data. *Trends in Cognitive Science, 10* 424-430.
- Nummenmaa L, Hietanen JK, Calvo MG & Hyona J (2011). Food catches the eye but not for everyone: A BMI-contingent attentional bias in rapid detection of nutriments. PLOS One, 6:e19215.
- Poldrack RA (2011). Inferring mental states from neuroimaging data: From reverse inference to large-scale decoding. *Neuron, 72*, 692-697.

- C Ravussin E & Bogardus C (2000). Energy balance and weight regulation: genetics versus environment. *British Journal of Nutrition, 83*, S17-20.
- Roefs A, Franssen S & Jansen A (2018). The dynamic nature of food reward processing in the brain. *Current Opinion in Clinical Nutrition and Metabolic Care, 21*, 444-448.
- Roefs A, Houben K & Werthmann J (2015). Desire for food and the power of mind. In W Hofmann & LF Nordgren (Eds.). *The Psychology of Desire* (pp. 323 – 346). New York: Guilford Press.
- Roefs A, Huijding J, Smulders FTY, Jansen, A & MacLeod CM (2015). Implicit measures of associations: A case of exaggerated promises? In G Brown & D Clarke (Eds.). *Cognitive Behavioral Assessment, Diagnosis and Case Formulation* (pp. 291 – 315). New York: Guilford Press.
- Roefs A & Jansen A (2002). Implicit and explicit attitudes toward high-fat foods in obesity. *Journal of Abnormal Psychology, 111,* 517-521.
- Roefs A et al. (2006). The environment influences whether high-fat foods are associated with palatable or with unhealthy. *Behaviour Research and Therapy, 44*, 715-736.
- Rothemund Y et al. (2007). Differential activation of the dorsal striatum by high-calorie visual food stimuli in obese individuals. *Neuroimage, 15*, 410-421.
- Smith ER & DeCoster J (2000). Dual-process models in social and cognitive psychology: Conceptual integration and links to underlying memory systems. *Personality and Social Psychology Review*, *4*, 108–131.
- Spanakis G, Weiss G, Boh B, Kerkhofs V & Roefs, A. (2016). Utilizing longitudinal data to build decision trees for profile building and predicting eating behavior. *Procedia Computer Science, 100*, 782-789.
- Spanakis G, Weiss G, Boh B & Roefs A (2016). Network analysis of Ecological Momentary Assessment data for monitoring and understanding eating behavior. *LNCS 9545*, 43-54.
- Strack F & Deutsch R (2004). Reflective and impulsive determinants of social behavior. *Personality and Social Psychology Review, 8*, 220-247.
- Volkow ND, Wang GJ, Tomasi D & Baler RD (2013). Obesity and addiction: neurobiological overlaps. *Obesity Reviews, 14*, 2-18.
- Wardle J (2009). Current issues and new directions in psychology and health: The genetics of obesity-What is the role for health psychology? *Psychology & Health, 24*, 997-1001.
- Werrij MQ et al. (2009). Adding cognitive therapy to dietetic treatment is associated with less relapse in obesity. *Journal of Psychosomatic Research, 67*, 315-324.
- Werthmann J, Jansen A. & Roefs A (2015). Worry or craving? A selective review of evidence for food-related attention biases in obese individuals, eating disorder patients, restrained eaters and healthy samples. *Proceedings of the Nutrition Society, 74*, 99-114.
- Werthmann J, Jansen A, Roefs A (2016). Make up your mind about food: A healthy mindset attenuates attention for high-calorie food in restrained eaters. *Appetite, 105*, 53-59.
- Werthmann J, Roefs A, Nederkoorn C, Mogg K, Bradley BP & Jansen A (2011). Can(not) take my eyes off it: Attention bias for food in overweight participants. *Health Psychology, 30*, 561-569.
- Yarkoni T, Poldrack RA, Nichols TE, Van Essen DC & Wager TD (2011). Large-scale automated synthesis of human functional neuroimaging data. *Nature Methods, 8*, 665-670.
- Yeshurun YS et al. (2017). Same story, different story: The neural representation of interpretive frameworks. *Psychological Science, 28*, 307-319.
- Zelissen PMJ et al. (2005). Effect of three treatment schedules of recombinant methionyl human leptin on body weight in obese adults: a randomised placebo-controlled trial. *Diabetes, Obesity and Metabolism, 7*, 755-761.
- Ziauddeen H, Faroqi S & Fletcher PC (2012). Obesity and the brain: how convincing is the addiction model? *Nature Reviews Neuroscience, 13*, 279-286.